

THE UNIQUE IMPACT OF INTUITIVE EATING
ON PHYSICAL INDICATORS OF HEALTH

By

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THE UNIQUE IMPACT OF INTUITIVE EATING
ON PHYSICAL INDICATORS OF HEALTH

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Abstract: Current obesity treatment strategies have made progress in addressing the obesity epidemic, but still obesity rates are not declining and follow-up studies sometimes present less-than-satisfying results (e.g., failure to achieve lasting treatment effects, development of eating pathology). As a response to these issues, intuitive eating (IE), a weight-neutral approach to obesity treatment, has emerged. The aim of this thesis is to investigate whether IE is related to multiple indicators of health independent of body mass index (BMI) among a diverse sample of adults. IE was measured with the Intuitive Eating Scale-2 (IES-2) and measured health indicators included blood pressure, fasting glucose, fasting insulin, triglycerides, and total, LDL, and HDL cholesterol. A series of hierarchical linear regressions was performed in order to analyze the associations between total and subscale IE with health indicators after controlling for BMI and other relevant covariates. 248 adults (32 ± 14 years old, 73% female, 64% white) participated in the study. After adjusting for BMI no significant associations between IE (total or subscale scores) and health indicators were observed. Implications for the use of IE in obesity treatment/health promotion and areas of future research are discussed

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CHAPTER I

INTRODUCTION

The Obesity Epidemic: Statement of the Problem

Over 35% of American adults currently meet the Center for Disease Control's (CDC) criteria for being obese, and every state in America has an adult obesity prevalence of at least 20% [1, 2]. Rates are even higher in some minority populations, most notably with 54% of American Indians/Alaska Natives being obese [1, 3]. This epidemic is of great concern because obesity is related to multiple preventable negative health outcomes (e.g., diabetes mellitus, cardiovascular disease, stroke), poorer psychological health, and places a substantial financial burden on the American economy and individual citizens [4, 5].

Due to these considerable global, local, and individual costs, the obesity epidemic has garnered well-deserved attention. Current treatment approaches for obesity most often consist of behavioral weight loss (BWL) interventions, typically characterized by lifestyle intervention focused on decreasing calorie intake and increasing physical activity [6]. Evidence suggests that this treatment method is particularly effective for inducing short-term weight loss. Systematic reviews have shown an average initial (i.e., 6 month) weight loss of 8.5% of body weight, and that diet/exercise interventions produce significantly greater weight loss than wait-list control or usual care [7, 8]. At follow-up (i.e., three to four years) an average loss of 4% of initial body weight is maintained and diet/exercise interventions continue to outperform control [7, 8].

Weaknesses in Current Treatment Approaches: Is This the Best Answer to the Problem?

Though the current obesity treatment approach often leads to initial losses of weight, approximately 46% of lost weight is regained across studies [9]. Researchers have concluded that, across the board, weight loss maintenance is poor and individuals typically regain most, if not all, of the weight that they lose [10-13]. Therefore, the unique effects of repeated cycles of losing and gaining weight, or weight cycling, on health must be considered. Weight cycling is defined as “the repeated loss and regain of body weight” [14]. Though there are notable limitations in the current literature, it appears that weight cycling may lead to metabolic shifts, more rapid adipose tissue growth, and increased risk of heart attack and stroke [15] though other literature suggests that weight cycling does not have substantial negative impact [16].

Though the primary target of BWL interventions is physical health, the treatment can affect many other aspects of individuals’ lives. In fact, a review of the literature revealed that dietary restraint – a tendency to consciously restrict food intake that is often promoted in BWL calorie reduction plans – is associated with a host of negative psychosocial factors [17]. Specifically, higher levels of self-reported dietary restraint are associated with impairments in general psychological functioning, excessive body and shape concerns, and problematic food-related attitudes and behaviors. With these findings in mind, it may not be surprising that dieting has also frequently been linked to more extreme disordered eating behaviors [17-27].

Over the past thirty years dieting has been found to be a significant risk factor for the development of disordered eating [17, 19-22]. Additionally, comorbidity between overweight/obesity and eating disorder (ED) – particularly binge eating disorder (BED) and/or bulimia nervosa (BN) – often occurs [24, 26, 27]. In spite of this common comorbidity between obesity and ED, the treatment approaches for the two categories are somewhat in conflict with

each other [28, 29]. We must begin to consider that individuals seeking treatment for obesity may concurrently have disordered relationships with food, and be cognizant that our current treatment approaches may contribute to the development of problematic eating behaviors in otherwise healthy individuals.

In sum, behavioral weight loss approaches have significant weaknesses; long-term maintenance of weight loss is rare, and negative psychosocial impacts are possible. Though it may seem a radical departure from the current standard of care, the problems with current treatment methods have led some researchers to consider a new paradigm that is less focused on weight [30, 31]. This alternative, weight-neutral approach to obesity treatment does not assume that weight loss is a necessary precursor to health improvement. Some evidence suggests that improvements in physiological markers of health (e.g., blood pressure, cholesterol) can be found in the absence of weight loss [32-35].

There are a variety of interventions utilizing a weight-neutral approach, or non-diet paradigm, that have been studied [33]. Though they have differences, all of these approaches are similar in that they include a focus on eating based on internal cues rather than dietary prescriptions. There is an objectively defined construct that allows for the measurement of this aspect of weight-neutral approaches, and it is known as intuitive eating (IE) [36].

Intuitive Eating: A New Answer to the Problem

IE is an approach to eating that is based on listening to the body's physiological cues and minimizing dietary restraint [36]. Currently, IE is conceptualized with four facets:

a) unconditional permission to eat (PERM), b) eating for physical rather than emotional reasons (PHYS), c) reliance on hunger and satiety cues (REL), and d) body-food choice congruence (CON). These facets are described in detail in Table 1.

IE has been positively associated with a broad range of psychological and behavioral factors [17, 37, 38]. Specifically, IE has been found to have a positive relationship with general psychological well-being, body image and self-esteem, and pleasure from eating. Additionally, IE has displayed a negative relationship with preoccupation with food and disordered eating. However, in order to promote IE as a health-promoting alternative to dieting, it must be shown to have beneficial effects on physical health as well.

Table 1

<i>Description of Intuitive Eating Scale-2 (IES-2) Subscales</i>	
Intuitive Eating Subscale (Scale Abbreviations)	Description
Unconditional Permission to Eat (UPE; PERM)	Giving oneself permission to eat whenever and whatever food is desired.
Eating for Physical Reasons (EPR; PHYS)	Consuming food in response to hunger, and not in response to emotions.
Reliance on Hunger and Satiety Cues (RHSC; REL)	Both being aware of and trusting internal signals of hunger and fullness.
Body-Food Choice Congruence (B-FCC; CON)	Choosing foods that are pleasurable and provide good fuel for the body.

IE is consistently negatively associated with body mass index (BMI) in cross-sectional studies [38-44], and weight-neutral interventions are often associated with either a maintenance of or decrease in weight [32, 35, 45-49]. However, fewer studies include additional biological variables and available results are mixed, though there is some evidence that IE is associated with various physical health indicators. For example, Hawks and colleagues found that individuals high in IE displayed lower BMI, higher high-density lipoprotein (HDL) cholesterol, lower triglycerides, and lower cardiovascular risk than individuals low in IE [42]. Bacon and colleagues [32] conducted a randomized clinical trial evaluating a non-diet intervention and a

typical diet intervention and found that the non-diet group showed sustained improvements in total cholesterol, LDL cholesterol, and systolic BP [32]. However, it is important to note that neither of these studies controlled for BMI when investigating the relationships between IE and health.

Other investigations have been conducted to evaluate non-dieting interventions as well. Recent reviews of these studies have suggested that these interventions may positively impact blood pressure and blood lipids [34, 50, 51]. Specific findings across the literature include improvements in levels of total cholesterol [32, 35, 47, 48], HDL cholesterol [46], LDL cholesterol [32, 35, 47, 48], triglycerides [35], systolic BP [32, 35, 48, 49, 52], and diastolic BP [46, 48, 49, 52]. However, there is also at least one published study that did not observe an effect for each one of these variables [32, 35, 46-48, 53, 54], and none of these studies considered the impact of BMI on the IE-health relationships. Taken together, these findings suggest that IE and the new weight-neutral paradigm *may* lead to both psychological and physical benefits that are maintained in the long-term.

Current Study

Despite the above evidence of potential benefits, several gaps exist in the literature and are the focus of this thesis. Significant weaknesses in the current literature exist, including non-representative samples, inconsistent study designs, and a lack of foundational research. The objective of the current study is to observe the unique baseline association of IE with physical health indicators in a diverse sample of adults, independent of weight status. The primary aims of this thesis are to a) determine whether IE is associated with greater overall health in a diverse population and b) evaluate whether this relationship remains after adjusting for BMI. The

exploratory aim of this thesis is to determine whether there are differences in the relationships between IE, BMI, and health based on age, gender, race, and/or obesity status.

Based on a critical evaluation of published studies it is hypothesized that IE will be negatively related to total cholesterol and LDL cholesterol. However, due to the current status of the literature (i.e., highly mixed) no *a priori* hypotheses can be made of IE's relation to HDL cholesterol, systolic blood pressure, diastolic blood pressure, fasting glucose, or fasting insulin. We predict that BMI will be associated with all health outcome variables, such that higher BMI would be related to values indicating poorer health (e.g., higher BMI, higher blood pressure; higher BMI, lower HDL cholesterol, etc.). Additionally, as consistent with the current literature, we predict that IE would be related to a decreased BMI [51]. This will be the first study to directly evaluate the impact of IE on health indicators after adjusting for the effects of BMI. Consequently, we make no *a priori* hypothesis regarding the extent to which IE is uniquely associated with the dependent variables, above and beyond body mass index.

CHAPTER II

METHOD

Overview

Data used in the current study came from baseline data collected from larger, ongoing projects, including two trials of behavioral weight loss in obese adults: *Cognitive and Self-Regulatory Mechanisms of Obesity Study (COSMOS; K23DK103941-01A1)* and *Pilot of Weight Reduction in Underserved Populations (POWER-UP; U54GM104938)* as well as community and laboratory conducted studies. All measures utilized in the present study are located in Appendix B.

Participants

Participants included obese adults from the community enrolled in a weight loss trial (i.e., COSMOS, POWER-UP), community members of all weight statuses, and college students enrolled at Oklahoma State University (OSU). We deliberately utilized data from these multiple sources to ensure that we achieved a sample diverse with regards to age, gender, race, and/or obesity status. Compensation was provided to all participants. COSMOS and POWER-UP participants received monetary compensation, community members received small gift cards, and OSU students received course credit. All participants were recruited with convenience sampling, through emails to OSU faculty and staff, the Psychology Department Research Participation System (SONA), or flyers advertising community data collection at local businesses. Inclusion criteria for this study were: a) age ≥ 21 years old and ≤ 65 years old and b) speak English fluently. Exclusion criteria were as follows: a) individuals out of the stated age

range, b) those who were currently pregnant, c) history of a neurological disorder, and/or d) non-English speaking. We estimated that a sample size of at least 240 participants would be needed based on the power analysis reported below.

Measures

Intuitive eating (IES-2). The IES-2 [55] is a 23-item self-report instrument that measures an individual's tendency to eat based on his/her body's internal cues. The scale provides a total IE score, along with subscale scores for the four facets of IE. The subscales are a) Unconditional Permission to Eat (PERM), b) Eating for Physical Rather than Emotional Reasons (PHYS), c) Reliance on Hunger and Satiety Cues (REL), and d) Body-Food Choice Congruence (CON). Responses range from 1 (Strongly Disagree) to 5 (Strongly Agree), and items are averaged to provide the total and subscale scores, with higher scores representing higher levels of IE.

The IES-2 has previously displayed good reliability and validity in both women and men [55]. Specifically, Cronbach's coefficient alphas for internal consistency were .87 and .89 for women and men, respectively. Further, previously reported Cronbach's coefficient alphas for the subscales ranged from .82 (PERM, women) to .92 (PHYS, women) for the four subscales. In the current study the IES-2 total score displayed good reliability as well (women $\alpha = .84$; men $\alpha = .79$). For this sample, the PHYS, REL, and CON subscales Cronbach's coefficient alphas were in the acceptable ($\alpha = .76$, PHYS, women) to good range ($\alpha = .89$, CON, men and women). However, the PERM subscale displayed lower reliability ($\alpha = .58$, men; $\alpha = .68$, women).

Health indicators. Multiple measures that have been shown to be related to physical health were examined in this study. Specifically, these measures are predictive of negative health

outcomes commonly associated with obesity (e.g., hypertension, type 2 diabetes, cardiovascular disease).

Body mass index. BMI was measured continuously as a function of participants' height and weight (kg/m^2). Participants' height (cm) and weight (kg) was measured directly by research personnel using research-grade scales: Tanita scale (TANITA Body Fat Analyzer Model TBF-105 K930599) or seca scale (Model 813). BMI was calculated using a standard formula: $(\text{weight} \times 0.45 / (\text{height} \times 100)^2)$.

Blood pressure. Systolic and diastolic blood pressures (mmHg) were measured with an electronic sphygmomanometer by research personnel. When multiple readings were available, the average of the first three readings was taken.

Metabolic factors. Metabolic factors consisted of glucoregulatory (i.e., fasting glucose, fasting insulin) and lipid (i.e., triglycerides, total, LDL, HDL cholesterol) factors. Fasting glucose levels were obtained in one of three ways. Community members' and OSU students' fasting glucose was measured with a glucometer by research personnel. POWER-UP participants' fasting glucose was measured with a home glucometer and self-reported. COSMOS participants' fasting glucose was obtained through clinic blood draws. Fasting insulin, total cholesterol, HDL cholesterol, LDL cholesterol, and triglyceride values were obtained through a clinic blood draw in a fasting state. These values were only available for a subset of participants given the higher costs of venous blood draws.

Demographic factors and covariates. Participants completed a questionnaire assessing demographic variables, including gender, age, race, and education level. These demographic variables were included as covariates in the data analysis.

Procedure

All data was collected from the baseline visit of the larger, ongoing studies across multiple settings. All participants involved in this thesis signed informed consent documents approved by the Oklahoma State University Institutional Review Board (IRB) and were adequately compensated. IE was measured during the baseline visit via self-report using the IES-2 [55]. Demographic factors and covariates (i.e., age, gender, race, education level) were self-reported. BMI was objectively measured by trained research personnel. Blood pressure readings were obtained by trained research personnel with an electronic sphygmomanometer. Fasting glucose levels were obtained in one of three ways: a) in the laboratory by trained research personnel with a glucometer, b) measured at home via glucometer and self-reported, or c) through clinic blood draw. All glucose measurements were taken following a fast of at least eight hours. All remaining measures (i.e., fasting insulin, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides) were measured via fasting clinic blood draw. These measures are only available for the subset of participants enrolled in the COSMOS study. After the completion of data collection all data was cleaned and analyzed. Statistical analyses consisted of a series of hierarchical linear regressions performed in SPSS. A detailed data analysis plan can be seen below.

Power Analysis for Primary Analyses. A power analysis was conducted using G*Power version 3 [56] to determine the sample size needed for the present study. Effect sizes were calculated from pilot data on 65 participants via post-hoc power analyses for all dependent variables: systolic blood pressure ($f^2 = .065$), diastolic blood pressure ($f^2 = .051$), total cholesterol ($f^2 = .0014$), HDL cholesterol ($f^2 = .044$), LDL cholesterol ($f^2 = .033$), triglycerides ($f^2 = .037$), fasting glucose ($f^2 = .004$), and fasting insulin ($f^2 = .131$). In order to be conservative in

estimating the required sample size, the final power analyses were based on the smallest meaningful effect (i.e., $\geq .02$) for each category of outcome variables (i.e., blood pressure, metabolic factors). [57] Results indicated that a sample size of 156 people are needed to have an 80% chance of detecting a small effect ($f^2 = .051$; diastolic blood pressure) of blood pressure at the 5% level (one-tailed). Additionally, it was found that a sample size of 240 people is needed to have an 80% chance of detecting a small effect ($f^2 = .033$; LDL cholesterol) of the metabolic factors at the 5% level (one-tailed).

Data Cleaning. All data were reviewed prior to analysis to assure completion and adequacy based on the assumptions for hierarchical linear regression. First, it was confirmed that all data was within the possible range for each variable. Any out-of-range data was cross checked with raw data and, if necessary, corrected to assure accuracy. Additionally, normality of the distribution was evaluated through skew and kurtosis. Data was considered normal if skewness was less than 3.0 and kurtosis was less than 10.0 [58]. Further, a score was considered an outlier if its standardized value was greater than or equal to 3.3. Any outlier that impacted the normality of the data was removed; all other outliers were retained. Missing data was imputed via within-person within-subscale mean imputation when $\leq 20\%$ of subscale responses were missing.

Data Analysis. A series of hierarchical linear regression analyses was performed to evaluate the ability of IE to predict health indicators when adjusting for BMI. The independent variable was IE scores at baseline and the dependent variables were systolic and diastolic blood pressure, total, HDL, and LDL cholesterol, triglycerides, fasting glucose, or fasting insulin (all measured at baseline). A separate analysis was conducted to evaluate IE on each of the outcome variables. For all analyses, covariates included age, gender, race, and education level. These covariates allowed for the conclusion of whether IE is associated with health indicators

independent of demographic factors. All covariates were entered in Step 1 and IE was entered in Step 2. Additionally, BMI was entered in Step 3 in order to observe whether it impacted the relationship between IE and health indicators. A partial Bonferroni correction was performed according to SISA guidelines [59], based on eight analyses with an alpha level of 0.05 and an average correlation of 0.12 between dependent variables. The calculation revealed a corrected alpha level of 0.012. Therefore, this alpha level (i.e., 0.012) was used as the criteria for statistical significance for all primary outcome variables. Of note, given that study participants came from several settings, stratified analyses were performed for outcomes present in all study groups (i.e., SBP, DBP, fasting glucose) to assure that there were not different patterns of results among the college/community and obese/treatment-seeking samples.

To investigate the exploratory aim of the study (e.g., differences based on demographic factors), moderation analyses were performed for all statistically significant predictors. Specifically, for each independent variable (e.g., total, subscale IE) that displayed a significant association with health, a moderation analysis was used to observe whether there was an interaction effect of IE with age, sex, race/education, and/or obesity status on the relevant dependent variable.

CHAPTER III

RESULTS

Participants

Participants were included in the final sample if they met all eligibility criteria and had complete data for all demographic and intuitive eating measures. Three individuals were excluded due to ineligibility (i.e., age > 65) and 31 individuals were excluded due to missing demographic or intuitive eating values. The final sample consisted of 248 adults who were 32.2 ± 14.3 years old, 73% female, and 64% White. Fifteen percent of the final sample were of an American Indian or Alaskan Native background. Participants had a mean BMI of 30.4 ± 7.6 kg/m², and all weight categories were represented (BMI range 18.2-55.3 kg/m²). These data suggest that we were able to recruit a sample diverse in respect to age, race, and obesity status. However, females were overrepresented, as is typical for studies of obesity and/or weight loss. Mean values for other collected biomarkers (e.g., blood pressure, glucose) were within the normal range. At the group level, participants displayed IE scores of 3.3 ± 0.5 on average for the 1-5 scale. Generally, total IE displayed a moderate-to-large effect size in association with BMI ($r = -.448$), as did three of its subscales (PHYS $r = -.371$, REL $r = -.341$, CON $r = -.393$). PERM displayed no correlation with BMI ($r = -.033$). Detailed demographic and descriptive data can be found in Table 2.

Body Mass Index

As planned, BMI was included in all models examining IE total or IE subscales as related to health indicators. It was predicted that BMI would be significantly associated with all health

indicators across the two series of models (i.e., models using total IE vs. models using IE subscales). Although minor variations existed for the regression coefficients and p-values across these two series of models, the relationship between BMI and the health indicators followed a similar pattern whether IE total or the IE subscales were the predictors. Consequently, the following detailed statistics are from the regression analyses with total IE. BMI displayed significant associations with DBP ($\beta = .220, p = .003$) and fasting insulin ($\beta = .370, p = .003$). The relationships between BMI and the remaining outcome variables (i.e., SBP, fasting glucose, triglycerides, total, HDL, LDL cholesterol) were non-significant (see Tables 3, 5, and 7) but were in the expected directions.

Primary Results

Overview. Primary analyses consisted of a series of hierarchical linear regressions meant to evaluate the unique relationship of IE with various markers of physical health. A detailed analysis plan can be found above. In general, observed associations of IE with health indicators did not differ before and after the inclusion of BMI in the model. Therefore, the results discussed in the following section are from Step 3 of the regressions, following the inclusion of BMI, unless otherwise specified. Regression coefficients for primary outcome variables were considered to be statistically significant if they met the Bonferroni-corrected significance criterion of $\alpha = .012$. Stratified analyses confirmed no differences between study populations (i.e., community/college vs. obese/treatment-seeking), therefore all results presented below are from the aggregate sample.

Intuitive Eating & Health Indicators. Associations of total IE scores (TOT) with SBP, DBP, fasting glucose, fasting insulin, triglycerides, total cholesterol, LDL cholesterol, and HDL cholesterol – following the inclusion of covariates and BMI – were first examined (see Tables 3,

5, and 7). IE subscales – PERM, PHYS, REL, and CON – were then analyzed to determine their unique associations with the previously stated health indicators (see Tables 4, 6, and 8).

Blood Pressure. Though there was a significant association between TOT and DBP in Step 2 ($\beta = -.191, p = .003$), TOT showed a non-significant relationship with blood pressure levels (SBP $\beta = -.077, p = .252$; DBP $\beta = -.123, p = .070$) after the inclusion of BMI (see Table 3). With regards to the IE subscales: Prior to considering BMI (i.e., Step 2), CON displayed a significant association with DBP ($\beta = -.225, p = .004$). However, no IE subscales displayed significant associations with blood pressure (i.e., SBP or DBP) following the inclusion of BMI (see Table 4). Likewise, effect sizes of IE with BP were in the range of small effects or below ($f^2 = .01-.06$; see Tables 3 and 4).

Glucoregulatory Factors. TOT was not significantly related to levels of fasting glucose ($\beta = .048, p = .497$) or fasting insulin ($\beta = -.077, p = .492$) (see Table 5). In addition, no IE subscale displayed significant associations with fasting glucose or fasting insulin following the inclusion of BMI (see Table 6). In line with above, effect sizes for these analyses reflected small effects or below ($f^2 = .00-.09$; see Tables 5 and 6).

Lipid Factors. The omnibus tests of TOT's relationship to triglycerides ($F(6, 67) = .862, p = .527$) and LDL cholesterol ($F(6, 67) = .479, p = .821$) were non-significant; therefore, the associated regression coefficients were not interpreted (see Table 7). Prior to the inclusion of BMI on Step 3, the omnibus test of TOT's relationship to total cholesterol was significant ($F(5, 68) = 2.444, p = .043$) (i.e., Step 2), though TOT did not display a significant association with the outcome ($\beta = .119, p = .300$). Following the inclusion of BMI on Step 3, the omnibus test for total cholesterol was no longer significant ($F(6, 67) = 2.021, p = .075$). Lastly, the relationship between TOT and HDL cholesterol was non-significant as well ($\beta = .113, p = .289$).

With regards to the IE subscales, analyses of triglycerides ($F(9, 64) = .604, p = .789$), total cholesterol ($F(9, 64) = 1.467, p = .180$), and LDL cholesterol ($F(9, 64) = .569, p = .817$) displayed non-significant omnibus tests; therefore, interpretation for these outcomes was not continued. Additionally, no IE subscales displayed significant associations with HDL cholesterol following the inclusion of BMI. These results can be seen in Table 8. Again, effects sizes of IE with lipid factors were in the small range or below ($f^2 = .00-.10$; see Tables 7 and 8).

In line with the data analysis plan presented above, as none of the primary relationships between IE and health were significant no follow-up analyses to investigate interaction effects were performed.

Table 2.

Participant Characteristics

	Max N = 248 M (SD) or N (%)
Demographics & History	
Age	32.18 (14.29)
Gender (female)	182 (73.4)
Education Level	
Some High School	7 (2.8)
High School	31 (12.5)
Some College	76 (30.6)
Bachelor's Degree	70 (28.2)
Graduate or Professional Degree	64 (25.8)
Race/Ethnicity	
African American	13 (5.2)
American Indian/Alaska Native	37 (14.9)
Asian/Pacific Islander	7 (2.8)
Caucasian	159 (64.1)
Hispanic	7 (2.8)
Other	5 (2.0)
Multiple	20 (8.1)
Biomarkers & Obesity Indicators	
BMI (kg/m ²)	30.41 (7.56)
Blood Pressure (mmHg)	
SBP	116.06 (13.15)
DBP	74.64 (10.14)
Fasting Glucose (mg/dL)	95.68 (26.83)
Fasting Insulin (mIU/L)	17.91 (9.60)
Triglycerides (mg/dL)	128.59 (65.43)
Total Cholesterol (mg/dL)	188.85 (36.86)
LDL Cholesterol (mg/dL)	113.08 (32.97)
HDL Cholesterol (mg/dL)	51.15 (15.16)
Intuitive Eating (IES-2)	
Total (TOT)	3.27 (0.51)
Unconditional Permission to Eat (PERM)	3.21 (0.68)
Eating for Physical Rather than Emotional Reasons (PHYS)	3.24 (0.84)
Reliance on Hunger and Satiety Cues (REL)	3.29 (0.77)
Body-Food Choice Congruence (CON)	3.41 (0.89)

Note. BMI = body mass index; DBP = diastolic blood pressure;

IES-2 = Intuitive Eating Scale-2; SBP = systolic blood pressure

Table 3.

Associations between Total IE and Blood Pressure

	Systolic Blood Pressure (n=243)			Diastolic Blood Pressure (n=243)		
<i>Step 1</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>
Demographics ^a	.210	--	15.808*	.138	--	9.493*
<i>Step 2</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>
	.217	.007	2.147	.169	.031	8.830*
	<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>	
TOT IE	-.092	.144		-.191	.003*	
<i>Step 3</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>
	.218	.001	.426	.198	.030	8.745*
	<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>	
Age	.216	.002*		.201	.005*	
Sex	-.397	<.001*		-.132	.032*	
Race/Ethnicity	-.062	.301		-.010	.869	
Education	-.103	.102		-.071	.262	
BMI	.048	.515	<i>f</i>²	.220	.003*	<i>f</i>²
TOT IE						
	-.077	.252	.01 (no effect)	-.123	.070	.04 (small effect)

Note: For omnibus statistics and covariates *Significant at $p < 0.05$

For primary outcomes (bolded) *Significant at $p < .012$

BMI = body mass index; TOT IE = Intuitive Eating Scale-2 total score

^aFor parsimony, the specific beta coefficients for the demographic variables (age, sex, race/ethnicity, education) are presented for Step 3 only.

Table 4.

Associations between IE Subscales and Blood Pressure

	Systolic Blood Pressure (n=243)			Diastolic Blood Pressure (n=243)		
	R^2	ΔR^2	ΔF	R^2	ΔR^2	ΔF
Step 1						
Demographics ^a	.210	--	15.808*	.138	--	9.493*
Step 2						
	R^2	ΔR^2	ΔF	R^2	ΔR^2	ΔF
	.233	.023	1.730	.189	.051	3.672*
	β	p		β	p	
PERM	-.138	.041		-.116	.094	
PHYS	-.044	.524		-.031	.665	
REL	.081	.243		-.025	.725	
CON	-.118	.121		-.225	.004*	
Step 3						
	R^2	ΔR^2	ΔF	R^2	ΔR^2	ΔF
	.234	.001	.285	.213	.024	7.158*
	β	p		β	p	
Age	.231	.001*		.199	.006*	
Sex	-.399	<.001*		-.115	.066	
Race/Ethnicity	-.053	.377		-.020	.743	
Education	-.093	.145		-.046	.475	
BMI	.040	.594		.202	.008*	
PERM	-.135	.046		-.102	.136	
PHYS	-.036	.614		.011	.878	
REL	.081	.239	.03 (small effect) ^b	-.021	.763	.06 (small effect) ^b
CON	-.110	.158		-.182	.021	

Note: For omnibus statistics and covariates *Significant at $p < 0.05$

For primary outcomes (bolded) *Significant at $p < .012$

BMI = body mass index; CON = Intuitive Eating Scale-2 Body-Food Choice Congruence subscale score;

PERM = Intuitive Eating Scale-2 Unconditional Permission to Eat subscale score;

PHYS = Intuitive Eating Scale-2 Eating for Physical Rather than Emotional Reasons subscale score;

REL = Intuitive Eating Scale-2 Reliance on Hunger and Satiety Cues subscale score.

^aFor parsimony, the specific beta coefficients for the demographic variables (age, sex, race/ethnicity, education) are presented for Step 3 only.

^bThe f^2 effect size is change in variance when all IE subscales were entered. It is not specific to one subscale.

Table 5.

Associations between Total IE and Glucose Regulatory Factors

	Fasting Insulin (n=72)			Fasting Glucose (n=212)		
<i>Step 1</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>
Demographics ^a	.095	--	1.756	.242	--	16.527*
<i>Step 2</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>
	.114	.019	1.437	.243	.001	.304
TOT IE	<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>	
	-.141	.235		.037	.582	
<i>Step 3</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>
	.231	.117	9.860*	.244	.001	.270
	<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>	
Age	-.129	.271		.468	<.001*	
Sex	-.173	.131		.094	.140	
Race/Ethnicity	-.097	.391		.057	.370	
Education	.206	.075		-.100	.133	
BMI	.370	.003*	<i>f</i>²	.041	.604	<i>f</i>²
TOT IE	-.077	.492	.02 (small effect)	.048	.497	.00 (no effect)

Note: For omnibus statistics and covariates *Significant at $p < 0.05$

For primary outcomes (bolded) *Significant at $p < .012$

BMI = body mass index; TOT IE = Intuitive Eating Scale-2 total score

^aFor parsimony, the specific beta coefficients for the demographic variables (age, sex, race/ethnicity, education) are presented for Step 3 only.

Table 6.

Associations between IE Subscales and Glucoregulatory Factors

		Fasting Insulin (n=72)			Fasting Glucose (n=212)		
Step 1		R^2	ΔR^2	ΔF	R^2	ΔR^2	ΔF
	Demographics ^a	.095	--	1.756	.242	--	16.527*
Step 2		R^2	ΔR^2	ΔF	R^2	ΔR^2	ΔF
		.173	.078	1.489	.244	.002	.120
		β	p		β	p	
	PERM	-.206	.119		.034	.635	
	PHYS	-.256	.066		.007	.926	
	REL	.128	.363		.024	.743	
	CON	.022	.886		.005	.959	
Step 3		R^2	ΔR^2	ΔF	R^2	ΔR^2	ΔF
		.267	.094	7.941*	.245	.001	.233
		β	p		β	p	
	Age	-.166	.178		.469	<.001*	
	Sex	-.223	.070		.092	.160	
	Race/Ethnicity	-.067	.560		.059	.371	
	Education	.156	.205		-.098	.150	
	BMI	.337	.006*		.039	.630	
	PERM	-.144	.256		.037	.604	
	PHYS	-.193	.146		.013	.861	
	REL	.113	.396		.025	.739	
	CON	.033	.818		.013	.873	

Note: For omnibus statistics and covariates *Significant at $p < 0.05$

For primary outcomes (bolded) *Significant at $p < .012$

BMI = body mass index; CON = Intuitive Eating Scale-2 Body-Food Choice Congruence subscale score;

PERM = Intuitive Eating Scale-2 Unconditional Permission to Eat subscale score;

PHYS = Intuitive Eating Scale-2 Eating for Physical Rather than Emotional Reasons subscale score;

REL = Intuitive Eating Scale-2 Reliance on Hunger and Satiety Cues subscale score.

^aFor parsimony, the specific beta coefficients for the demographic variables (age, sex, race/ethnicity, education) are presented for Step 3 only.

^bThe f^2 effect size is change in variance when all IE subscales were entered. It is not specific to one subscale.

Table 7.

Associations between Total IE and Lipid Factors

	Triglycerides (n=74)			Total Cholesterol (n=74)			LDL Cholesterol (n=74)			HDL Cholesterol (n=74)		
<i>Step 1</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>
Demographics ^a	.058	--	1.058	.139	--	2.779*	.038	--	.688	.251	--	5.780* 1
<i>Step 2</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>
	.063	.005	.382	.152	.014	1.092	.041	.003	.179	.272	.021	1.945
TOT IE	<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>	
	-.074	.539		.119	.300		.051	.673		.147	.168	
<i>Step 3</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>
	.072	.009	.623	.153	.001	.0.69	.041	.000	.019	.299	.027	2.613
	<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>	
Age	-.078	.534		-.049	.684		-.101	.430		.220	.047*	
Sex	.011	.931		.373	.002*		.156	.217		.448	<.001*	
Race/Ethnicity	-.137	.263		-.008	.946		.016	.895		.035	.742	
Education	-.156	.212		-.029	.804		-.007	.953		.123	.259	
BMI	.101	.433	<i>f</i>²	-.032	.793	<i>f</i>²	.018	.892	<i>f</i>²	-.180	.111	<i>f</i>²
TOT IE	-.055	.654	.01	.113	.337	.02	.054	.662	.00	.113	.289	.03
	(no effect)			(small effect)			(no effect)			(small effect)		

Note: For omnibus statistics and covariates *Significant at $p < 0.05$

For primary outcomes (bolded) *Significant at $p < .012$

BMI = body mass index; TOT IE = Intuitive Eating Scale-2 total score

^aFor parsimony, the specific beta coefficients for the demographic variables (age, sex, race/ethnicity, education) are presented for Step 3 only.

Table 8.

Associations between IE Subscales and Lipid Factors

	Triglycerides (n=74)			Total Cholesterol (n=74)			LDL Cholesterol (n=74)			HDL Cholesterol (n=74)		
<i>Step 1</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>
Demographics ^a	.058	--	1.058	.139	--	2.779*	.038	--	.688	.251	--	5.780*
<i>Step 2</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>
	.067	.010	.167	.170	.031	.611	.074	.035	.622	.317	.066	1.578
	<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>	
PERM	.046	.733		.111	.381		-.066	.625		.243	.038	
PHYS	-.053	.708		-.039	.775		-.119	.405		.048	.697	
REL	-.063	.659		.064	.637		.154	.282		-.067	.585	
CON	.018	.908		.161	.274		.090	.560		.234	.082	
<i>Step 3</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>	<i>R</i>²	<i>ΔR</i>²	<i>ΔF</i>
	.078	.011	.458	.171	.001	.081	.074	.000	.016	.338	.021	1.981
	<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>		<i>β</i>	<i>p</i>	
Age	-.072	.596		-.073	.568		-.140	.302		.198	.087	
Sex	.008	.951		.325	.013*		.100	.456		.409	.001*	
Race/Ethnicity	-.146	.247		-.004	.971		.041	.747		.019	.860	
Education	-.146	.286		-.054	.679		-.052	.706		.103	.374	
BMI	.116	.387	<i>f</i>²	-.036	.777	<i>f</i>²	-.017	.901	<i>f</i>²	-.159	.164	<i>f</i>²
PERM	.070	.612		.104	.426		-.069	.617		.211	.074	
PHYS	-.031	.833	.01 (no effect)	-.046	.741	.04 (small effect)	-.122	.404	.04 (small effect)	.016	.895	.10 (small effect)
REL	-.069	.628		.066	.630		.155	.283		-.058	.634	
CON	.023	.881		.160	.282		.090	.566		.227	.089	

Note: For omnibus statistics and covariates *Significant at $p < 0.05$

For primary outcomes (bolded) *Significant at $p < .012$

BMI = body mass index; CON = Intuitive Eating Scale-2 Body-Food Choice Congruence subscale score;

PERM = Intuitive Eating Scale-2 Unconditional Permission to Eat subscale score;

PHYS = Intuitive Eating Scale-2 Eating for Physical Rather than Emotional Reasons subscale score;

REL = Intuitive Eating Scale-2 Reliance on Hunger and Satiety Cues subscale score.

^aFor parsimony, the specific beta coefficients for the demographic variables (age, sex, race/ethnicity, education) are presented for Step 3 only.

^bThe f^2 effect size is change in variance when all IE subscales were entered. It is not specific to one subscale.

CHAPTER IV

DISCUSSION

The objective of this study was to observe the unique association of IE with physical health indicators, independent of weight status. Overall, the observed results suggest that IE does not have a unique relationship with physical health indicators in a diverse sample of adults after adjusting for BMI. Due to the highly mixed state of previous literature in this area, the only specific *a priori* hypotheses that were made were that BMI would be related to all health indicators and IE would be negatively related to total cholesterol and LDL cholesterol.

BMI displayed a significant positive association with DBP and fasting insulin in the current sample. In contrast to hypothesized results, it did not display significant associations with SBP, fasting glucose, triglycerides, total, LDL, or HDL cholesterol. Though they did not reach significance, each of these relationships was in the hypothesized direction. These findings were likely influenced by limitations in power and variable measurement, which are discussed in detail below.

Results showed no significant associations between IE (total or subscale scores) and total or LDL cholesterol prior to or after the inclusion of BMI. Significant associations between total IE and body-food choice congruence (CON) with DBP were observed prior to the consideration of BMI, however, no significant associations between total or subscale IE scores with blood pressure (i.e., SBP, DBP), glucoregulatory (i.e., fasting glucose, fasting insulin), or lipid (i.e., total, LDL, HDL cholesterol, triglycerides) factors were observed after adjusting for the role of objective weight status.

The present findings are consistent with some of the previous literature, though there are numerous contradictory findings. For example, for total and LDL cholesterol, previous findings would suggest that there likely is a relationship between IE and health, with four studies finding an effect of IE for both total and LDL cholesterol [32, 35, 47, 48]. Only two studies found no association with total cholesterol [42, 54] and one study found no association with LDL cholesterol [42]. Approximately half of studies evaluating the impact of IE on blood pressure have found beneficial effects as well [32, 35, 46-49, 52-54]. Likewise, the results of studies evaluating HDL cholesterol and triglycerides in relation to IE have also been split with approximately 50% finding an association and 50% failing to find an association [32, 35, 42, 46-48]. However, no previous work has found a significant relationship between IE and fasting glucose, and the impact of IE on fasting insulin has not previously been evaluated [42, 46-48].

The previous study with a design most similar to the current study (i.e., cross-sectional) was an analysis of differences in health indicators between college females who were high and low in IE [42]. Both studies investigated the presence of a baseline relationship between IE and health indicators commonly associated with obesity. Hawks and colleagues concluded that individuals high in IE displayed better health than those low in IE, as observed by lower BMI, lower triglycerides, higher HDL cholesterol, and lower Total/HDL cholesterol ratio. An important difference between these two studies is that Hawks and colleagues measured IE with the Intuitive Eating Scale [60], created by Hawks et al., while the present study used the Intuitive Eating Scale-2 [55], created by Tylka and Kroon Van Diest. These measures are designed to measure the same construct but have not been directly compared to allow for confidence in their convergent validity. Beyond differences in measurement, it is possible that the discrepancy between these results and those observed in the current study are due to Hawks et al.'s extreme

groups design, which likely increased the chance of detecting an effect by maximizing the variability in IE scores. Further, it is not clear whether Hawks and colleagues controlled for relevant covariates (e.g., age, sex, race/ethnicity, and BMI). It is likely that Hawks et al.'s failure to control for BMI and demographic factors in the IE-health relationship as well as their use of a non-diverse study sample (i.e., college females) were significant contributors to the discrepant findings between the two studies.

More generally, the current study differed from previous literature in two important ways: study design and participant characteristics. The current study was a cross-sectional analysis of baseline associations, while many of the previous studies were experimentally-designed interventions. While experimental studies are generally considered a stronger design, one would expect that an effect observed experimentally would typically be present cross-sectionally as well. Therefore, the aim of this thesis was to increase knowledge of IE's basic relationships with health, in order to serve as a foundation for continuing to refine longitudinal IE studies that could justify IE's utility as an alternative/adjunctive obesity treatment. Unfortunately, the results of this study did not support the presence of a cross-sectional IE-health relationship that is independent of the effects of BMI.

There are various factors that may explain the differing results between this study and intervention studies. First, it is possible that previous interventions displayed significant improvements in health following a non-diet intervention via mechanism(s) of action other than IE (e.g., social support, depression). Specifically, intervention studies have varied in the comparison groups utilized, and some have been more strictly controlled than others, but it could be possible that participants' health benefited from factors such as membership in a treatment group or nutritional education. Additionally, it could be that significant effects of IE are observed

in intervention studies due to the ability to observe effects over time. Perhaps IE is acting indirectly on health through its well-established association with psychosocial factors, some of which (e.g., depression) have been shown to impact physical health [37, 61]. If this is true, it could be difficult to detect the relationship without longitudinal data and/or mediation analyses. Further, it may be that the change in IE is more important than baseline levels of IE. If so, that would explain why improvements in health are sometimes observed in response to an increase in IE behaviors. All of these are methodological reasons why the current findings may have differed from previous studies based on research design; however, differences in participant characteristics could be a factor as well.

The current study contained greater participant diversity in terms of age, race/ethnicity, and weight status than many of the previous studies. By increasing representation of all ages, minority populations, and both normal and overweight/obese individuals, this study sought to gain a better understanding of the relationships between IE and health indicators in the overall population, while maintaining power to investigate moderating factors. It seems plausible that the significant effects in previous literature could be due to contextual effects, such as the effect of IE on health indicators being present specifically within an obese population. However, sensitivity analyses were performed when possible which revealed no differences between the community/college and obese/treatment-seeking populations in the current study.

Implications

The results of this study have a variety of implications for the clinical application of IE and future research in this area. Many researchers have called for a paradigm shift in obesity treatment, in which the IE/weight-neutral approach is utilized over traditional BWL consisting of diet and exercise. Because IE often does not lead to a decrease of weight [51], in order for it to

be a viable approach to obesity treatment it must improve health independently of BMI. This study did not reveal a unique relationship of IE with any of a number of health indicators commonly associated with negative consequences of obesity. This finding, in conjunction with the absence of consistent support of a unique IE-health relationship in previous literature, does not support continued efforts to move the field toward a decreased emphasis on weight loss in obesity treatment.

Though IE's potential as a stand-alone obesity treatment was not supported by the current findings, there are still multiple ways in which IE might serve as an effective health promotion technique. Firstly, IE – and three of its subscales – displayed small to moderate negative correlations with BMI in the present sample, which has previously been found in numerous studies [34, 51]. This suggests that even if IE does not help to improve health status independent of weight loss, having an intuitive approach to eating is associated with having a lower baseline weight. Therefore, IE may be very well suited as an obesity prevention technique. Focusing on encouraging young people to focus on their body's physical needs over emotional, social, or environmental cues for eating may help to decrease the incidence of obesity development. Based on these findings, future research on the utility of an IE intervention for obesity prevention is warranted.

Additionally, apart from acting as a stand-alone obesity treatment, IE may still have utility for individuals who have developed overweight/obesity. Previous research has consistently supported various psychological and behavioral benefits of IE, and, specifically, in some areas (e.g., eating behaviors, self-esteem) that it appears dieting may have the potential to be psychologically detrimental [17, 37]. Future research might investigate whether IE could serve as pre-treatment to BWL, specifically for individuals who may have experienced

psychological or behavioral detriments of dieting in the past (e.g., chronic dieting, disordered eating). Perhaps IE could help these individuals to establish a healthier relationship with food and foster more positive feelings about the self, which would likely increase their ability to be adhere to a healthful eating plan and to be successful at losing and maintaining weight.

Lastly, IE may be able to play a role in improving the maintenance of weight losses. Many studies of IE-based interventions have revealed an attenuation of weight gain within overweight and obese populations [34, 51]. This finding, along with the previously mentioned psychological and behavioral benefits of IE, supports additional research on the impact of IE for individuals who have successfully improved their health via weight loss and are attempting to establish a sustainable pattern of adaptive eating.

Limitations

In discussing the implications of these findings on IE utility, various limitations of the current study must be considered. Firstly, a cross-sectional study design has some limitations by nature. Because all observations are made at a single time point it is impossible to obtain information regarding dynamic relationships between the study variables. Further, without randomization and experimenter manipulation it is difficult to rule out confounding factors that may have influenced the results. Though statistical techniques were used to account for known covariates, there could be unobserved or unknown factors affecting the results. These limitations inhibit the ability to draw causal conclusions from the given data. However, the cross-sectional design was appropriate for the research question this study sought to answer.

In addition to limitations of the cross-sectional design, the study contained some limitations in variable measurement. Though the IES-2 has been previously validated and generally displayed good reliability in the current sample, the unconditional permission to eat

subscale showed reliability in the poor to questionable range. This suggests that the PERM subscale may not have been performing in a way that would provide a reliable measure of the desired construct in the current sample. Therefore, conclusions regarding the results of this subscale may not be valid. Further, as a self-report instrument the IES-2 is prone to some general error. It may be difficult for individuals to accurately report their typical eating behaviors and responses could be influenced by social desirability, though all participants were assured of confidentiality and encouraged to answer honestly. Additionally, there were some weaknesses in the measurement of blood pressure and fasting glucose. Multiple measurements over various days/times yield the most valid measures of these biomarkers, though in the current study they were approximated with a lower sampling frequency over a single visit. This measurement technique was appropriate given the study design and financial resources and in that it did not cause participants undue burden. However, more sophisticated measures of these indicators (e.g., ambulatory home blood pressure readings over several days and use of the oral glucose tolerance test) would likely yield more valid and reliable results of these indicators.

Further, due to limited resources not all outcome measures were available for all participants. Specifically, blood pressure (i.e., SBP and DBP) and fasting glucose were the only dependent variables available for the entire study sample. The remaining dependent measures – lipid factors and fasting insulin – were only available for a subset of the sample, and this subset was all obese, treatment-seeking individuals. To investigate effects of sample characteristics stratified analyses were performed when possible (i.e., SBP, DBP, fasting glucose) and no differences in results were observed between community/college and obese, treatment-seeking individuals. However, it is impossible to conclude with certainty whether the remaining outcomes were or were not influenced by these demographic factors. Further, due to the smaller

sample size available for lipid factors and fasting insulin these outcomes may have been underpowered, though the effect sizes for these measures do not suggest medium-strong effects are present ($f^2 < 0.02$).

Conclusions

In sum, the current study revealed no association between IE and health indicators (i.e., blood pressure, metabolic factors) that was independent of the effects of BMI. As IE has not been previously shown to decrease weight, these findings are in conflict to the recent call for a revision to obesity treatment standards in which an emphasis is placed on weight-neutral approaches. However, these findings do not negate the previously discussed weaknesses in current obesity treatment methods and benefits of IE outside of cardiovascular and metabolic health. There are a variety of ways in which IE may be useful in health promotion, and future research is warranted to continue working to discover how IE may play a role in improving our fight against the obesity epidemic and its negative psychosocial risk factors and concomitants.

REFERENCES

1. Ogden, C.L., et al., *Prevalence of Obesity Among Adults and Youth: United States, 2011-2014*. NCHS Data Brief, 2015(219): p. 1-8.
2. Control, C.f.D. *Adult Obesity Prevalence Maps*. Overweight and Obesity 2017.
3. Levi, J., et al., *State of Obesity: Better Policies for a Healthier America: 2015*. 2015: Trust for America's Health.
4. Control, C.f.D. *Adult Obesity Causes and Consequences*. Overweight and Obesity 2016 August 15, 2016 [cited 2017 May 30].
5. Dixon, J.B., *The effect of obesity on health outcomes*. Molecular and cellular endocrinology, 2010. **316**(2): p. 104-108.
6. Jensen, M.D., et al., *2013 AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults*. Circulation, 2014. **129**(25 suppl 2): p. S102-S138.
7. Franz, M.J., et al., *Weight-loss outcomes: a systematic review and meta-analysis of weight-loss clinical trials with a minimum 1-year follow-up*. Journal of the American Dietetic Association, 2007. **107**(10): p. 1755-1767.
8. Dombrowski, S.U., A. Avenell, and F.F. Sniecott, *Behavioural interventions for obese adults with additional risk factors for morbidity: systematic review of effects on behaviour, weight and disease risk factors*. Obesity facts, 2010. **3**(6): p. 377-396.
9. Barte, J., et al., *Maintenance of weight loss after lifestyle interventions for overweight and obesity, a systematic review*. Obesity Reviews, 2010. **11**(12): p. 899-906.

10. Miller, W.C., *How effective are traditional dietary and exercise interventions for weight loss?* Medicine and Science in Sports and Exercise, 1999. **31**(8): p. 1129-1134.
11. Fletcher, S.W., et al., *Methods for voluntary weight loss and control. NIH Technology Assessment Conference Panel.* Annals of internal medicine, 1992. **116**(11): p. 942.
12. Goodrick, G.K., W.S. Poston, and J.P. Foreyt, *Methods for voluntary weight loss and control: update 1996.* Nutrition, 1996. **12**(10): p. 672-676.
13. Mann, T., et al., *Medicare's search for effective obesity treatments: diets are not the answer.* American Psychologist, 2007. **62**(3): p. 220.
14. WIN and NIDDK, *Weight cycling.* Updated March 2006.. ed. 2006, Bethesda, MD]: Bethesda, MD : National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health.
15. Strohacker, K., K.C. Carpenter, and B.K. Mcfarlin, *Consequences of weight cycling: An increase in disease risk?* International journal of exercise science, 2009. **2**(3): p. 191.
16. Mehta, T., et al., *Impact of weight cycling on risk of morbidity and mortality.* Obesity Reviews, 2014. **15**(11): p. 870-881.
17. Hawks, S.R., H.N. Madanat, and H.S. Christley, *Psychosocial associations of dietary restraint: Implications for healthy weight promotion.* Ecology of food and nutrition, 2008. **47**(5): p. 450-483.
18. Haines, J. and D. Neumark-Sztainer, *Prevention of obesity and eating disorders: a consideration of shared risk factors.* Health education research, 2006. **21**(6): p. 770-782.
19. Jacobi, C., et al., *Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy.* Psychol Bull, 2004. **130**(1): p. 19-65.

20. Neumark-Sztainer, D., et al., *Obesity, disordered eating, and eating disorders in a longitudinal study of adolescents: how do dieters fare 5 years later?* Journal of the American Dietetic Association, 2006. **106**(4): p. 559-568.
21. Stice, E., C.N. Marti, and S. Durant, *Risk factors for onset of eating disorders: Evidence of multiple risk pathways from an 8-year prospective study.* Behaviour research and therapy, 2011. **49**(10): p. 622-627.
22. Polivy, J. and C.P. Herman, *Dieting and bingeing. A causal analysis.* The American psychologist, 1985. **40**(2): p. 193.
23. Boutelle, K., et al., *Weight control behaviors among obese, overweight, and nonoverweight adolescents.* J Pediatr Psychol, 2002. **27**(6): p. 531-40.
24. Hudson, J.I., et al., *The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication.* Biological psychiatry, 2007. **61**(3): p. 348-358.
25. Neumark-Sztainer, D., et al., *Sociodemographic and personal characteristics of adolescents engaged in weight loss and weight/muscle gain behaviors: who is doing what?* Prev Med, 1999. **28**(1): p. 40-50.
26. Neumark-Sztainer, D., et al., *Weight-related concerns and behaviors among overweight and nonoverweight adolescents: implications for preventing weight-related disorders.* Arch Pediatr Adolesc Med, 2002. **156**(2): p. 171-8.
27. Flament, M.F., et al., *Weight Status and DSM-5 Diagnoses of Eating Disorders in Adolescents From the Community.* Journal of the American Academy of Child & Adolescent Psychiatry, 2015. **54**(5): p. 403-411.e2.
28. Irving, L.M. and D. Neumark-Sztainer, *Integrating the Prevention of Eating Disorders and Obesity: Feasible or Futile?* Preventive Medicine, 2002. **34**(3): p. 299-309.

29. Neumark-Sztainer, D., *Can we simultaneously work toward the prevention of obesity and eating disorders in children and adolescents?* International Journal of Eating Disorders, 2005. **38**(3): p. 220-227.
30. Bacon, L. and L. Aphramor, *Weight science: evaluating the evidence for a paradigm shift.* Nutrition journal, 2011. **10**(1): p. 1.
31. Bombak, A., *Obesity, health at every size, and public health policy.* Journal Information, 2014. **104**(2).
32. Bacon, L., et al., *Size acceptance and intuitive eating improve health for obese, female chronic dieters.* Journal of the American Dietetic Association, 2005. **105**(6): p. 929-936.
33. Cadena-Schlam, L. and G. López-Guimerà, *Intuitive eating: an emerging approach to eating behavior.* Nutrición hospitalaria, 2014. **31**(3): p. 995.
34. Schaefer, J.T. and A.B. Magnuson, *A review of interventions that promote eating by internal cues.* Journal of the Academy of Nutrition and Dietetics, 2014. **114**(5): p. 734-760.
35. Bacon, L., et al., *Evaluating a "non-diet" wellness intervention for improvement of metabolic fitness, psychological well-being and eating and activity behaviors.* International Journal of Obesity, 2002.
36. Tribole, E. and E. Resch, *Intuitive eating: a recovery book for the chronic dieter: rediscover the pleasures of eating and rebuild your body image.* 1995.
37. Bruce, L.J. and L.A. Ricciardelli, *A systematic review of the psychosocial correlates of intuitive eating among adult women.* Appetite, 2016. **96**: p. 454-472.
38. Denny, K.N., et al., *Intuitive eating in young adults. Who is doing it, and how is it related to disordered eating behaviors?* Appetite, 2013. **60**: p. 13-19.

39. Madden, C.E., et al., *Eating in response to hunger and satiety signals is related to BMI in a nationwide sample of 1601 mid-age New Zealand women*. Public health nutrition, 2012. **15**(12): p. 2272-2279.
40. Smith, T. and S.R. Hawks, *Intuitive eating, diet composition, and the meaning of food in healthy weight promotion*. American Journal of Health Education, 2006. **37**(3): p. 130-136.
41. Tylka, T.L., R.M. Calogero, and S. Daniélsdóttir, *Is intuitive eating the same as flexible dietary control? Their links to each other and well-being could provide an answer*. Appetite, 2015. **95**: p. 166-175.
42. Hawks, S., et al., *The relationship between intuitive eating and health indicators among college women*. Journal of Health Education, 2005. **36**(6): p. 331-336.
43. Schoenefeld, S.J. and J.B. Webb, *Self-compassion and intuitive eating in college women: Examining the contributions of distress tolerance and body image acceptance and action*. Eating Behaviors, 2013. **14**(4): p. 493-496.
44. Tanco, S., W. Linden, and T. Earle, *Well-being and morbid obesity in women: A controlled therapy evaluation*. International Journal of Eating Disorders, 1998. **23**(3): p. 325-339.
45. Leblanc, V., et al., *Impact of a Health-At-Every-Size intervention on changes in dietary intakes and eating patterns in premenopausal overweight women: results of a randomized trial*. Clinical Nutrition, 2012. **31**(4): p. 481-488.

46. Carroll, S., E. Borkoles, and R. Polman, *Short-term effects of a non-dieting lifestyle intervention program on weight management, fitness, metabolic risk, and psychological well-being in obese premenopausal females with the metabolic syndrome*. Applied Physiology, Nutrition, and Metabolism, 2007. **32**(1): p. 125-142.
47. Mensinger, J.L., et al., *A weight-neutral versus weight-loss approach for health promotion in women with high BMI: A randomized-controlled trial*. Appetite, 2016.
48. Rapoport, L., M. Clark, and J. Wardle, *Evaluation of a modified cognitive-behavioural programme for weight management*. International journal of obesity, 2000. **24**(12): p. 1726.
49. Mellin, L., M. Croughan-Minihane, and L. Dickey, *The Solution Method: 2-year trends in weight, blood pressure, exercise, depression, and functioning of adults trained in development skills*. Journal of the American Dietetic Association, 1997. **97**(10): p. 1133-1138.
50. Clifford, D., et al., *Impact of non-diet approaches on attitudes, behaviors, and health outcomes: a systematic review*. Journal of nutrition education and behavior, 2015. **47**(2): p. 143-155. e1.
51. Van Dyke, N. and E.J. Drinkwater, *Review article relationships between intuitive eating and health indicators: literature review*. Public health nutrition, 2014. **17**(08): p. 1757-1766.
52. Hawley, G., et al., *Sustainability of health and lifestyle improvements following a non-dieting randomised trial in overweight women*. Preventive medicine, 2008. **47**(6): p. 593-599.

53. Ciliska, D., *Evaluation of two nondieting interventions for obese women*. Western Journal of Nursing Research, 1998. **20**(1): p. 119-135.
54. Steinhardt, M.A., J.R. Bezner, and T.B. Adams, *Outcomes of a traditional weight control program and a nondiet alternative: a one-year comparison*. The Journal of psychology, 1999. **133**(5): p. 495-513.
55. Tylka, T.L. and A.M. Kroon Van Diest, *The Intuitive Eating Scale–2: Item refinement and psychometric evaluation with college women and men*. Journal of Counseling Psychology, 2013. **60**(1): p. 137.
56. Faul, F., et al., *G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences*. Behavior research methods, 2007. **39**(2): p. 175-191.
57. Cohen, J., *Statistical power analysis for the behavioral sciences*. 1977.
58. Kline, R.B., *Principles and practice of structural equation modeling*. 1998.
59. Utienbroek, D.G., *SISA binomial*. 1997: Southhampton: DG Utienbroek.
60. Hawks, S., R.M. Merrill, and H.N. Madanat, *The intuitive eating scale: Development and preliminary validation*. American Journal of Health Education, 2004. **35**(2): p. 90-99.
61. Hare, D.L., et al., *Depression and cardiovascular disease: A clinical review*. European Heart Journal, 2014. **35**(21): p. 1365-1372.
62. Dombrowski, S.U., et al., *Long term maintenance of weight loss with non-surgical interventions in obese adults: systematic review and meta-analyses of randomised controlled trials*. 2014.
63. Control, C.f.D. *Defining Adult Overweight and Obesity*. Overweight and Obesity 2016 June 16, 2016 [cited 2017 May 11, 2017].

64. Panel, N.T.A.C., *Methods for voluntary weight loss and control*. Annals of Internal Medicine, 1993. **119**(7 pt 2): p. 764-770.
65. Ng, M., et al., *Global, regional, and national prevalence of overweight and obesity in children and adults during 1980-2013: a systematic analysis for the Global Burden of Disease Study 2013*. Lancet, 2014. **384**(9945): p. 766-81.
66. Singh, A.S., et al., *Tracking of childhood overweight into adulthood: a systematic review of the literature*. Obesity reviews, 2008. **9**(5): p. 474-488.
67. Flegal, K.M., et al., *Trends in obesity among adults in the united states, 2005 to 2014*. JAMA, 2016. **315**(21): p. 2284-2291.
68. Flegal, K.M., et al., *Association of all-cause mortality with overweight and obesity using standard body mass index categories: a systematic review and meta-analysis*. Jama, 2013. **309**(1): p. 71-82.
69. Peeters, A., et al., *Obesity in adulthood and its consequences for life expectancy: a life-table analysis*. Annals of internal medicine, 2003. **138**(1): p. 24-32.
70. Imes, C.C. and L.E. Burke, *The obesity epidemic: the USA as a cautionary tale for the rest of the world*. Current epidemiology reports, 2014. **1**(2): p. 82-88.
71. Lehnert, T., et al., *Economic costs of overweight and obesity*. Best practice & research Clinical endocrinology & metabolism, 2013. **27**(2): p. 105-115.
72. Tribole, E. and E. Resch, *Intuitive eating: A revolutionary program that works*. 2012: Macmillan.
73. Wing, R.R. and S. Phelan, *Long-term weight loss maintenance*. The American journal of clinical nutrition, 2005. **82**(1): p. 222S-225S.

74. Anderson, J.W., et al., *Long-term weight-loss maintenance: a meta-analysis of US studies*. The American journal of clinical nutrition, 2001. **74**(5): p. 579-584.
75. Control, C.f.D. *Losing Weight*. Healthy Weight 2015.
76. Howard, B.V., et al., *Low-fat dietary pattern and weight change over 7 years: the Women's Health Initiative Dietary Modification Trial*. Jama, 2006. **295**(1): p. 39-49.
77. Howard, B.V., et al., *Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial*. Jama, 2006. **295**(6): p. 655-666.
78. Wadden, T.A., et al., *Four - Year Weight Losses in the Look AHEAD Study: Factors Associated With Long - Term Success*. Obesity, 2011. **19**(10): p. 1987-1998.
79. Turk, M.W., et al., *Randomized clinical trials of weight-loss maintenance: A review*. The Journal of cardiovascular nursing, 2009. **24**(1): p. 58.
80. Obesity, M., and Nutrition Institute, *Course in Obesity Medicine: Treating Obesity 2017*, P. Sumithran, Editor. 2017. p. 1307.
81. O'Hara, L. and J. Gregg, *The war on obesity: a social determinant of health*. Health Promot J Austr, 2006. **17**(3): p. 260-263.
82. Tylka, T.L., et al., *The weight-inclusive versus weight-normative approach to health: Evaluating the evidence for prioritizing well-being over weight loss*. Journal of obesity, 2014. **2014**.
83. Robison, J., *Health at every size: toward a new paradigm of weight and health*. Medscape General Medicine, 2005. **7**(3): p. 13.
84. Tylka, T.L., *Development and psychometric evaluation of a measure of intuitive eating*. Journal of Counseling Psychology, 2006. **53**(2): p. 226.

85. Tribole, E. and E. Resch, *Intuitive Eating: A revolutionary program that works*. 2 ed. 2003.
86. Tylka, T.L. and J.A. Wilcox, *Are intuitive eating and eating disorder symptomatology opposite poles of the same construct?* Journal of Counseling Psychology, 2006. **53**(4): p. 474.
87. Gast, J. and S.R. Hawks, *Weight loss education: the challenge of a new paradigm*. Health education & behavior, 1998. **25**(4): p. 464-473.
88. Greene, G.W., et al., *Impact of an online healthful eating and physical activity program for college students*. American Journal of Health Promotion, 2012. **27**(2): p. e47-e58.
89. Hawks, S.R., et al., *Classroom approach for managing dietary restraint, negative eating styles, and body image concerns among college women*. Journal of American college health, 2008. **56**(4): p. 359-366.
90. Humphrey, L., D. Clifford, and M.N. Morris, *Health at every size college course reduces dieting behaviors and improves intuitive eating, body esteem, and anti-fat attitudes*. Journal of nutrition education and behavior, 2015. **47**(4): p. 354-360. e1.
91. Carbonneau, E., et al., *A Health at Every Size intervention improves intuitive eating and diet quality in Canadian women*. Clinical Nutrition, 2017. **36**(3): p. 747-754.
92. Anderson, L.M., et al., *Contributions of mindful eating, intuitive eating, and restraint to BMI, disordered eating, and meal consumption in college students*. Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity, 2016. **21**(1): p. 83-90.
93. Cole, R.E. and T. Horacek, *Effectiveness of the My Body Knows When intuitive-eating pilot program*. American journal of health behavior, 2010. **34**(3): p. 286-297.

94. Polivy, J. and C.P. Herman, *Undieting: A program to help people stop dieting*. International Journal of Eating Disorders, 1992. **11**(3): p. 261-268.
95. Bush, H.E., et al., *Eat for life: a work site feasibility study of a novel mindfulness-based intuitive eating intervention*. American Journal of Health Promotion, 2014. **28**(6): p. 380-388.
96. Mensinger, J.L., R.M. Calogero, and T.L. Tylka, *Internalized weight stigma moderates eating behavior outcomes in women with high BMI participating in a healthy living program*. Appetite, 2016. **102**: p. 32-43.
97. Andrew, R., M. Tiggemann, and L. Clark, *Predictors of intuitive eating in adolescent girls*. Journal of Adolescent Health, 2015. **56**(2): p. 209-214.
98. Augustus-Horvath, C.L. and T.L. Tylka, *The acceptance model of intuitive eating: a comparison of women in emerging adulthood, early adulthood, and middle adulthood*. Journal of Counseling Psychology, 2011. **58**(1): p. 110.
99. Avalos, L.C. and T.L. Tylka, *Exploring a model of intuitive eating with college women*. Journal of Counseling Psychology, 2006. **53**(4): p. 486.
100. Tylka, T.L. and K.J. Homan, *Exercise motives and positive body image in physically active college women and men: Exploring an expanded acceptance model of intuitive eating*. Body image, 2015. **15**: p. 90-97.

APPENDIX A

Extended Review of Literature

Overview

Obesity is a pressing concern in the United States, with over 35% of American adults currently meeting the Center for Disease Control's (CDC) criteria for being obese [1]. Further, every state in America has an adult obesity prevalence of at least 20% [2]. Obesity is related to multiple preventable negative health outcomes (e.g., diabetes mellitus, cardiovascular disease, stroke) and places a substantial financial burden on the American economy and individual citizens [4]. Over the past several decades there have been substantial efforts made to address the concern. However, obesity rates are not declining, and follow-up studies of traditional obesity interventions tend to present less-than-satisfying results (e.g., failure to achieve lasting treatment effects, development of eating pathology) [1, 30, 32, 62]. As a response to these issues, a weight-neutral approach to obesity treatment has emerged, which includes the intuitive eating construct [33]. While there is promising evidence of intuitive eating fostering a healthy relationship with food and promoting psychological well-being, there are many gaps in the literature on intuitive eating as it relates to physical health indicators. Specifically, it is unknown whether intuitive eating exerts positive effects on health, independent of an adult individual's weight status. Therefore, the aim of this thesis is to investigate whether intuitive eating is related to multiple indicators of health independent of body mass index among a diverse sample of adults.

The Obesity Epidemic: Statement of the Problem

Definition. Obesity is defined as a higher weight than what is considered healthy for a given height [63]. This is measured with the body mass index (BMI; kg/m^2), a continuous scale that takes into account both height and weight. A BMI of 18.5 to less than 25 kg/m^2 is considered normal weight and a BMI of 25.0 to less than 30 kg/m^2 is considered overweight. A person is considered obese if his or her BMI is 30 kg/m^2 or greater. Obesity is further defined based on three distinct categories. Class 1 obesity includes BMI 30 to < 35, class 2 obesity includes BMI 35 to < 40, and Class 3 obesity includes BMI 40 or greater. [63].

Causes/Risk Factors. There are many factors that lead to the development of obesity [64]. Broadly, these factors can be summarized with a biopsychosocial model that incorporates an individual's biological and psychological make-up, behavior, and environment [4]. Most commonly obesity is thought to be caused by particular behavioral patterns. In fact, a lifestyle that involves more calories taken in than expended will often lead to obesity. Behaviors associated with this lifestyle include the over-consumption of food and low levels of physical activity. However, the environment is also highly influential in many individuals' development of an obesogenic lifestyle. Many societies today have high-calorie food readily available and lack opportunities to engage in physical activity. Further, an individual's genetics can interact with environmental stimuli to increase the risk for obesity. Individuals who are genetically prone to higher levels of hunger or slower metabolisms may be more strongly affected by their behaviors or environment. Further, some diseases (e.g., polycystic ovarian syndrome), or their treatments (e.g., steroids), lead to an increased risk of developing obesity [4]. Through all of these identified mechanisms and many yet to be discovered, obesity has become a widespread epidemic in America and around the world.

Prevalence. Globally, approximately 37% of adults are overweight or obese [65]. In America, over 36% of adults are obese and an additional 36% of adults are overweight [1]. The equally high prevalence of overweight is important because studies have shown that overweight often progresses into obesity and has even been called “pre-obesity” [66]. The prevalence of obesity is slightly higher among American women (38%) than men (34%), and is highest in individuals aged 40-59 (40%). Further, there are substantial differences in obesity rates based on race and ethnicity. Rates are lowest in non-Hispanic Asian adults (11.7%) and highest in American Indians/Alaska Natives (54%) [1, 3]. Further, particularly high rates can be seen in non-Hispanic black women (56.9%) [1].

Trends. Recently, overall adult and child obesity rates in America have begun to level off. There was no significant change in the total number of American adults with obesity between 2011-2012 and 2013-2014 [1]. However, the prevalence of obesity in women did significantly increase from 2005 to 2014 [67]. Though rates of obesity are highest among minority groups, these populations are underrepresented in national surveys and there is not enough data available to adequately assess how obesity rates have changed over recent years in many of these groups (e.g., American Indians, Asian/Pacific Islanders) [3]. Specifically, increases were found in non-Hispanic white women, non-Hispanic black women, and Mexican American women [67]. Further, global rates of overweight and obesity have significantly increased from 1980 to 2013 [65].

Consequences. The obesity epidemic is of great concern because obesity is related to multiple preventable negative health outcomes and places a substantial financial burden on the American economy and individual citizens [4]. A meta-analysis by Flegal and colleagues revealed that obesity is associated with greater all-cause mortality compared to normal weight

[68]. Specifically, one study found that being obese decreased life expectancy by 5.8 years for males and 7.1 years for females [69]. Additional physical health impairments associated with obesity include cardiovascular disease, diabetes mellitus, obstructive sleep apnea, cancer, and general physical impairment [5, 70]. Further, obesity is associated with detriments to psychological health, including increased rates of depression, anxiety, and disordered eating, and decreases in body image, self-esteem, and quality of life [5].

Obesity is also linked to significant economic burden. Obese individuals spend 30% more worldwide and 43% more in America on healthcare than normal weight individuals [70, 71]. Further, the annual direct cost of obesity to the United States economy is estimated between 147 – 190 billion dollars, which is approximately 9.1 – 20.6% of total healthcare expenditures [71]. In addition, obesity is estimated to cost over 30 billion dollars in lost productivity (e.g., early death, absenteeism, disability) each year [70].

Due to these considerable global, local, and individual costs, the obesity epidemic has garnered well-deserved attention. Significant efforts have been made to address the obesity epidemic, and the increase in obesity prevalence has begun to slow. Current treatment approaches for obesity most often consist of behavioral weight loss (BWL) interventions, though there are alternative treatment approaches, including medications and surgical approaches for more severe levels of obesity (i.e., $\text{BMIs} \geq 35 \text{ kg/m}^2$). However, due to their widespread use and applicability to all classes of obesity, the discussion of current treatment approaches in this thesis will focus on behavioral weight loss interventions.

Current Obesity Treatment Approaches: Addressing the Problem

Current recommendations for the treatment of most obese individuals includes lifestyle intervention focused on decreasing calorie intake and increasing physical activity [6].

Specifically, the 2013 AHA/ACC/TOS Guideline for the Management of Overweight and Obesity recommends “comprehensive lifestyle intervention” (p. 111) as first-line treatment for all individuals with body mass index 30 kg/m^2 or greater, or 27 kg/m^2 or greater with associated comorbidities (e.g., hypertension) [6]. Ideally, the intervention should be in the form of a program - led by a trained professional - that includes a calorie deficit of approximately 500 calories per day, increased physical activity, and behavioral strategies to promote adherence to the plan. Each of these components should be uniquely tailored to the individual, but general recommendations include a range of 1200-1800 calories per day depending on body weight and a goal of reaching and maintaining 250 minutes of physical activity per week. Behavioral strategies include components of self-monitoring of food intake, physical activity, and body weight. See Table A.1, Column A for details regarding each general behavioral strategy in the current treatment paradigm.

Table A.1

Comparison of the Components of Current Behavioral Weight Loss Treatments to Intuitive Eating

General Behavioral Strategy	Column A: Current Treatment Approach	Column B: Intuitive Eating Approach
Self-Monitoring	Track daily caloric intake and weekly average Track minutes of PA Weigh approximately once per week	Carefully attend to physiological signs of hunger and fullness Carefully attend to the body's reactions to different foods
Method of Distribution	Attend regularly scheduled individual or group sessions with a trained professional <u>OR</u> receive electronically delivered intervention	No organized program. Guidelines in <i>Intuitive Eating: A Revolutionary Program that Works</i> [72]
Calorie Restriction	1200-1500 calories per day	No specific caloric limitations. Advised to engage in "gentle nutrition;" eat a variety of foods that make the body feel good and perform well.
Physical Activity Recommendations	250 minutes per week	Engage in pleasurable exercise for motives other than weight loss (e.g., increased energy level)
Weighing and Measuring Foods	Carefully weigh and measure all food in order to increase accuracy of tracking	Determine portion sizes based on internal signals of hunger and fullness.
Planning Meals and Times	Plan meals ahead of time and construct an environment readily available with healthy food choices	Plan meals ahead of time and construct an environment readily available with healthy food choices
Stimulus Control	Get rid of triggers for unhealthy eating behaviors. Remove access to unhealthy food, avoid restaurant eating, avoid people and places associated with unhealthy food choices.	Remove restrictions and grant self unconditional permission of when and what to eat.

Effectiveness of Current Obesity Treatments. Evidence suggests that current behavioral treatment methods are effective for treating adult obesity, particularly for inducing short-term weight loss. In 2007 Franz and colleagues performed a systematic review of obese adults' weight loss outcomes in clinical trials [7]. They included randomized clinical trials with weight loss as a primary outcome and a follow-up measure of at least one year. Weight loss intervention methods included advice, diet and/or exercise, meal replacements, pharmacological

treatments, and very-low-energy diets. A total of eighty studies were included, with seventeen utilizing diet and exercise as the primary intervention method. Relevant results showed that within the first six months of interventions using current treatment guidelines (i.e., diet and exercise) an average of 8.5% of initial body weight was lost. Generally, at 12 months weight loss had reached a plateau. Studies that measured additional time points showed an average loss of 4% of body weight was maintained at three- and four-year follow-ups.

More recently, Dombrowski and colleagues performed a similar systematic review of behavioral weight loss interventions in obese adults [8]. To be included, studies were required to target individuals with at least one additional risk factor for morbidity (e.g., type 2 diabetes) and utilize some combination of diet and exercise in the intervention. A total of 44 studies were included, with 27 evaluating combination diet-exercise interventions against wait-list control or usual care. Relevant results showed that these interventions led to more weight lost, on average, than comparison groups. The greatest difference in weight loss (3.5 kg) between intervention and comparison groups occurred at six months. At three-year follow-up intervention groups displayed an average weight loss of 2.6 kilograms more than comparison groups. Altogether, these results suggest that, generally, within the years following weight loss some weight is regained but a full rebound to baseline weight is not typically experienced [7, 8].

Weaknesses in Current Treatment Approaches: Is This the Best Answer to the Problem?

Physical. The above studies suggest that the current obesity treatment approach successfully leads to short-term weight loss, but also raise the question of whether it effectively produces long-term maintenance of weight loss. According to Wing and Phelan, only 20% of individuals who experience successful weight loss maintain their losses in the long term [73]. The issue of weight loss maintenance has been a concern for over two decades, but it has yet to

be sufficiently addressed [11, 12]. A meta-analysis of weight loss maintenance in the United States revealed that a decrease of only 2% body weight was maintained over a five-year follow-up [74], but current research has shown that a reduction of 5-10% of body weight is needed for health improvement [75]. Further, many BWL intervention studies do not include follow-up measures long enough to draw conclusions about ultimate weight outcomes.

One study that did provide a long-term follow-up of weight loss outcomes was the Women's Health Initiative (WHI), which has been deemed the "largest and longest randomized, controlled dietary intervention clinical trial" [30]. The WHI included a large-scale (n=48,835) clinical trial of a low-fat diet in a diverse sample of postmenopausal women [76]. Though participants in the intervention group were not instructed to reduce their overall calories, average caloric intake did decrease, and the given dietary guidelines were reflective of common obesity treatment recommendations [30]. As expected, the greatest weight loss was seen at the end of the first year. By the final follow-up measure, nearly eight years after the start of the study, participants in the intervention group were an average of 0.8 kilograms below their baseline weight [76]. This is equivalent to approximately 1.75 pounds of weight loss being maintained long-term. Further, no differences were found in the incidence of coronary heart disease, stroke, or cardiovascular disease between intervention and control groups, suggesting no long-term clinical benefit of the small amount of weight loss that was maintained [77].

Another strong study exemplar with a moderate follow-up measure was the Look AHEAD study [78]. The Look AHEAD study evaluated the impact of intentional weight loss via an Intensive Lifestyle Intervention (versus usual care) in over 5,000 individuals with type 2 diabetes. The greatest weight loss occurred at year one, and over the following three years weight was gradually regained. At the four-year follow-up participants had regained

approximately 50% of the weight they had lost. Further, it can be assumed that weight regain would have continued to increase if longer follow-up measures had been taken [13].

The results of the landmark WHI and Look AHEAD studies are not unique. A systematic review of lifestyle interventions for obesity with at least one-year follow-up found that approximately 46% lost weight was regained across studies [9]. Even BWL interventions targeted at increasing weight loss maintenance have shown limited success [79]. Researchers have concluded that – across the board, weight loss maintenance is poor and individuals typically regain most, if not all, of the weight that they lose [10-13]. One reason for poor weight loss maintenance is likely the metabolic adaptation that the body undergoes after losing weight [80]. The body biologically cannot tell the difference between therapeutic weight loss and starvation, so, in order to promote survival, it fights to regain any weight that is lost. Specific metabolic changes occur that make it easier to regain weight (e.g., reduced basal metabolic rate, increases in hunger hormones). Therefore, the unique effects of repeated cycles of losing and gaining weight, or weight cycling, on health must be considered.

Weight cycling is defined as “the repeated loss and regain of body weight” [14]. No specific amount of weight is required to be lost or regained in order to define a weight cycle. There is some debate over whether weight cycling is a cause for concern, but there is growing evidence that it may negatively impact health [14, 15]. In 2009 Strohacker and colleagues reviewed the literature to evaluate whether weight cycling increases disease risk. They recognized many limitations in the current body of literature (e.g., inconsistency in study design, population, and weight cycling definition) but concluded that based on available evidence it is likely that weight cycling leads to metabolic shifts, more rapid adipose tissue growth, and increased risk of heart attack and stroke [15]. At this point, the predominance of weight regain

and likelihood of adverse consequences of weight cycling cannot be ignored when prescribing weight loss as a primary obesity treatment.

Psychological. Though the primary target of BWL interventions is physical health, the treatment strategies can affect many other aspects of individuals' lives. In fact, a review of the literature revealed that dietary restraint is associated with a host of negative psychosocial factors [17]. While Hawks and colleagues did not specifically evaluate behavioral weight loss interventions, they defined dietary restraint as “the conscious effort to limit and control dietary intake, typically for the purpose of reducing or maintaining body weight,” which is highly reflective of the calorie restriction component of BWL treatments (p. 451). They reviewed 361 studies and found that higher levels of self-reported dietary restraint were associated with impairments in general psychological functioning, excessive body and shape concerns, and problematic food-related attitudes and behaviors. Specifically, lower levels of self-esteem and higher levels of depression, anxiety, and perceived stress were observed in high restraint individuals. Additionally, individuals high in dietary restraint tended to be highly concerned with body weight and related their self-worth to their body image and dieting success. Lastly, high levels of restraint were associated with food preoccupation, feelings of deprivation and cravings, guilt associated with eating, and a higher likelihood to overeat [17]. With these findings in mind, it may not be surprising that dieting has also frequently been linked to more extreme disordered eating behaviors [17-27].

Over thirty years ago Polivy and Herman [22] recognized that dieting is often followed by binge eating behaviors and concluded that this is a causal pathway. Since then it has been repeatedly found that dieting is a significant risk factor for the development of disordered eating [17, 19-21]. Even “healthful dieting,” as defined by “changing the way you eat to lose weight”

without engaging in unhealthful weight control behaviors (e.g., diet pills, laxatives, self-induced vomiting), was related to increased incidence of binge eating five years later in a prospective study of adolescent girls [20]. Further, it has been suggested that eating disorders (ED) – especially binge eating disorder (BED) and bulimia nervosa (BN) – share many risk factors with obesity, including dieting, media use, body dissatisfaction, and weight-related teasing [18].

The presence of shared risk factors for obesity and ED is supported by a frequent, but largely unrecognized, comorbidity between overweight/obesity and BED/BN. This comorbidity has been observed by multiple researchers in both adolescents and adults [24, 26, 27]. Neumark-Sztainer and colleagues (2002) found that 20% of overweight adolescent girls and 10% of overweight adolescent boys engage in binge eating, and Flament and colleagues (2015) found an association between bulimic disorders and obesity in adolescents. Further, Hudson and colleagues (2007) found that in adults in the National Comorbidity Survey Replication, BED was associated with BMI >40 kg/m².

In spite of the shared risk factors and common comorbidity between obesity and ED, the treatment approaches for the two categories are somewhat in conflict with each other [28, 29]. Treatment for obesity largely focuses on restricting food intake and careful self-monitoring, while treatment for BED and BN emphasize eliminating the restriction of foods and self-acceptance. Further, each treatment approach, when taken to an extreme, is highly reflective of the symptoms of the opposite disorder. For example, an excessive level of the dietary restriction recommended for obesity is observed in individuals with anorexia nervosa. Likewise, a complete abandoning of food restrictions can sometimes be seen in individuals with obesity. This observation reiterates the many previous findings that dieting behavior is often associated with the development of disordered eating habits [22, 24]. We must begin to consider that individuals

seeking treatment for obesity may concurrently have disordered relationships with food, and be cognizant that our current treatment approaches may contribute to the development of problematic eating behaviors in otherwise healthy individuals.

In sum, behavioral weight loss approaches have significant weaknesses; long-term maintenance of weight loss is rare, and negative psychosocial impacts are possible. Though it may seem a radical departure from the current standard of care, some researchers have concluded that the weight-loss approach may not be a useful treatment of obesity, and that it even may cause harm to individuals [81, 82]. Therefore, it has been questioned whether it is ethical to continue prescribing weight loss for the treatment of obesity [30, 31]. The problems with current treatment methods have led some researchers to consider a new paradigm that is less focused on weight [30, 31]. This alternative, weight-neutral approach to obesity treatment does not assume that weight loss is a necessary precursor to health improvement. Some evidence suggests that improvements in physiological markers of health (e.g., blood pressure, cholesterol) can be found in the absence of weight loss [32-35].

There are a variety of interventions utilizing a weight-neutral approach, or non-diet paradigm, that have been studied [33]. One common example that has resulted from this paradigm shift is the Health at Every Size (HAES) movement. The framework of HAES is based on acceptance in four realms: body shape and size, weaknesses of diet-based interventions, eating based on bodily signals, and psychosocial contributors to health [83]. The HAES model is one of the most well-defined weight-neutral treatments for obesity. Many other interventions have employed treatments that share similar goals, but are not systematically defined in the same way. However, all of these approaches include a focus on eating based on internal cues rather than dietary prescriptions. There is an objectively defined construct that allows for the

measurement of this aspect of weight-neutral approaches, and it is known as intuitive eating (IE) [36]. Therefore, the aims of this thesis will focus on the core tenet of IE, rather than weight-neutral obesity treatments broadly.

Intuitive Eating: A New Answer to the Problem

Definition of Intuitive Eating. Intuitive eating (IE), as first defined by Tribole and Resch, is an approach to eating that is based on listening to the body's physiological cues and minimizing dietary restraint [36]. The model was first objectively defined by Tylka [84] and has recently been revised and updated [55]. Currently IE is conceptualized with four facets: a) unconditional permission to eat (PERM), b) eating for physical rather than emotional reasons (PHYS), c) reliance on hunger and satiety cues (REL), and d) body-food choice congruence (CON). These facets are described in more detail in Table A.2. In addition to giving oneself permission to eat whenever and whatever food is desired [36], PERM is focused on removing labels of foods as “good” and “bad,” and minimizing the tendency to restrict certain foods or food groups [84]. PHYS is the ability to consume food in response to hunger rather than in response to emotions (e.g., stress, sadness, joy) [36]. REL is the ability to use the body's physiological signs of hunger and fullness, and trusting them to guide eating [36]. Body-food choice congruence refers to practicing “gentle nutrition,” [85] or choosing foods that both taste good and make the body perform well.

Table A.2

Description of Intuitive Eating Scale-2 (IES-2) Subscales

Intuitive Eating Subscale (Scale Abbreviations)	Description
Unconditional Permission to Eat (UPE; PERM)	Giving oneself permission to eat whenever and whatever food is desired.
Eating for Physical Reasons (EPR; PHYS)	Consuming food in response to hunger, and not in response to emotions.
Reliance on Hunger and Satiety Cues (RHSC; REL)	Both being aware of and trusting internal signals of hunger and fullness.
Body-Food Choice Congruence (B-FCC; CON)	Choosing foods that are pleasurable and provide good fuel for the body.

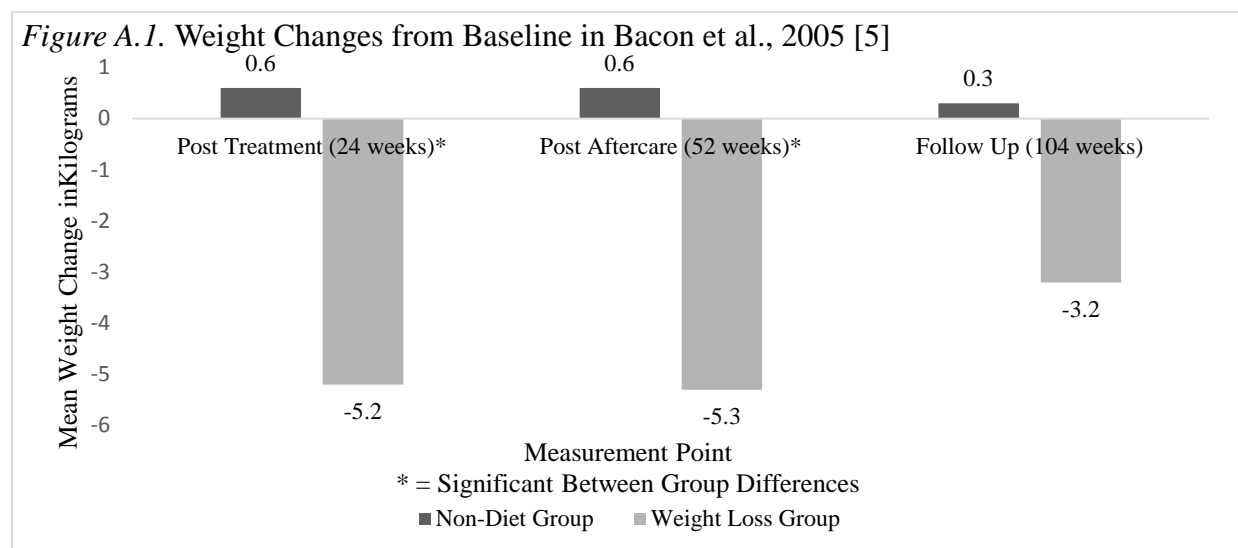
Taking these domains together, the intuitive eating approach can be conceptualized as eating based on physiological need, choosing foods that provide optimal fuel, and removing the restrictions of when and what to eat that are common in traditional diets. Details of this approach can be seen in Table A.1, Column B in order to contrast them with the traditional behavioral strategies. IE is meant to give individuals of any body weight a strategy to overcome the environmental and emotional eating patterns commonly fostered by the obesogenic environment. IE is considered an adaptive style of eating [33] and is an independent construct that cannot be explained as simply the absence of disordered eating behaviors or flexible dietary control [41, 86].

Since its emergence, IE has been positively associated with both psychological and physical factors [32-34, 37, 42]. Notably, no adverse effects of IE have been found [33]. This adaptive method may lead to overcoming the obesogenic environment and to fostering a natural, healthy relationship with food [87].

Psychological Correlates of Intuitive Eating. IE has been positively associated with a broad range of psychological and behavioral factors. Some examples include increased life satisfaction, increased positive and decreased negative affect, improved diet quality, and decreased chronic dieting [38, 39, 41, 43, 47, 88-91]. Further, IE is associated with many of the same psychological factors as dieting, but in the opposite direction. Whereas dieting is associated with guilt from eating, food preoccupation, and feelings of deprivation and cravings, IE is associated with pleasure from eating, decreased food preoccupation, and decreased levels of dietary restraint [32, 35, 40, 41, 89, 92-94]. As noted previously, dieting has been linked to increased disinhibition in eating and disordered eating behaviors, including binge eating and bulimia symptoms [17]. Intuitive eating has been inversely associated with the same behaviors [32, 35, 37-39, 41, 92, 94-96]. A high level of dietary restraint (i.e., dieting) is associated with increased depression, anxiety, and perceived stress; intuitive eating is linked to decreased depression, increased mindfulness, and increased distress tolerance [17, 32, 35, 43, 94, 95]. Dieting is related to excessive weight and shape concerns, while intuitive eating is frequently associated with improved body image and self-esteem – fostering a healthier view of the self and relationship with food [32, 35, 41, 43, 89, 90, 95, 97-100]. Further, many of these associations are sustained over time (i.e., up to two years), suggesting the ability to maintain benefits associated with a non-dieting approach to health. These findings suggest that intuitive eating may be an antidote to the many psychological consequences of dieting. However, in order to promote intuitive eating as an alternative to dieting, it must be shown to have beneficial effects on physical health as well.

Physical Correlates of Intuitive Eating. There is some evidence that intuitive eating is associated with physical health indicators. However, less is known about the impact of intuitive

eating on physical health compared to its impact on psychological well-being. Fewer studies include biological variables, and available results are mixed. Anthropometric variables – such as body weight and body mass index – are the most commonly measured category of physical health indicators in this body of literature. Intuitive eating is consistently negatively associated with body mass index in cross-sectional studies [38-44]. Further, even though weight loss is not a direct goal of intuitive eating (or non-dieting programs broadly), these interventions are often associated with either a maintenance of or decrease in weight [32, 35, 45-49]. When non-diet groups are compared with traditional weight loss (WL) interventions, the WL group often initially loses a greater amount of weight [35, 47, 48]. However, by the last follow-up measure the WL group has often begun to regain weight, whereas the non-diet group has continued to maintain or gradually lose weight, resulting in non-significant between group differences [32, 48]. An example of this progression is shown in Figure A.1.



Though the relationship between intuitive eating and body mass index is fairly well supported, research on intuitive eating’s association with other health indicators is sparse.

Currently, only one observational study of intuitive eating has evaluated multiple indicators of physical health. Hawks and colleagues [42] administered the Intuitive Eating Scale [60] to 205 college females in order to identify individuals high and low in intuitive eating. The final sample consisted of 15 high scorers and 17 low scorers. These extreme groups were compared on multiple health variables, including metabolic, cardiovascular, cardiorespiratory, and anthropometric factors. Results showed no difference in fasting glucose, total cholesterol, low-density lipoprotein (LDL) cholesterol, percent body fat, iron, and estimated VO₂ based on intuitive eating. However, it was found that individuals high in intuitive eating displayed lower body mass index, higher high-density lipoprotein (HDL) cholesterol, lower triglycerides, and lower cardiovascular risk than individuals low in intuitive eating [42].

There are also a small number of experimental studies that have evaluated the efficacy of non-diet programs on weight and health indicators. Bacon and colleagues [32] conducted a randomized clinical trial evaluating a non-diet (HAES) intervention and a typical diet intervention on a broad range of outcomes, including multiple indicators of physical health. Participants were 78 obese females, ages 30-45 years, with a history of chronic dieting. Treatment in both groups included six months of weekly group meetings, six months of monthly aftercare support, and a two-year follow-up. The HAES intervention had five specific treatment aspects: “body acceptance, eating behavior, nutrition, activity, and social support” [32]. Goals of treatment included separating feelings of self-worth from weight, learning to internally regulate eating rather than employing restrictive eating habits, and choosing food to support the body’s well-being. The diet intervention was “similar to most behavior-based weight-loss programs” (p. 930), and was based on the LEARN Program for Weight Control. Goals of treatment were to

restrict calorie intake, self-monitor food intake, and increase physical activity. Specific behavioral strategies to reach these goals were presented.

Results at the final follow-up measure revealed that the diet group initially showed improvements on some variables, but that many of these improvements were not sustained. However, the HAES group showed multiple sustained improvements [32]. Specifically, neither group showed significant decreases in weight by the follow-up measure, and groups were not different at follow-up in terms of weight or BMI. The diet group showed significant improvements in LDL cholesterol and systolic blood pressure (BP) at 1-year, but these improvements were not sustained at follow-up. The HAES group showed significant improvements in total cholesterol, LDL cholesterol, and systolic BP that were all sustained at follow-up. Both groups showed significant decreases in HDL cholesterol that were sustained at follow-up and no significant change in diastolic BP [32].

Other investigations have been conducted to evaluate various non-dieting interventions. Recent reviews of these studies have suggested that these interventions may positively impact blood pressure and blood lipids [34, 50, 51]. Specific findings across the literature include improvements in levels of total cholesterol [32, 35, 47, 48], HDL cholesterol [46], LDL cholesterol [32, 35, 47, 48], triglycerides [35], systolic BP [32, 35, 48, 49, 52], and diastolic BP [46, 48, 49, 52]. However, there is also at least one published study that did not observe an effect for each one of these variables [32, 35, 46-48, 53, 54]. Details of these studies can be seen in Table A.3, and a summary of their findings can be found in Table A.4.

Table A.3

Characteristics of Studies Evaluating Intuitive Eating and Multiple Physical Health Indicators

Study (first author, year)	Total sample	Study Design	Outcome Variables Assessed
Bacon, 2002; Bacon, 2005	78 female, Caucasian, obese chronic dieters aged 30-45	Randomized Clinical Trial Health at Every Size versus Diet Groups <ul style="list-style-type: none"> 6-month treatment, 2-year follow-up 	Body weight BMI Systolic BP Diastolic BP Total cholesterol HDL cholesterol LDL cholesterol Triglycerides
Carroll, 2007	31 pre-menopausal obese women with Metabolic Syndrome	Randomized Controlled Trial Non-dieting lifestyle intervention program (consistent with HAES and self-determination theory) versus Delayed-start control group <ul style="list-style-type: none"> 12-week treatment, 1-year follow-up 	Body weight BMI Systolic BP Diastolic BP HDL cholesterol Triglycerides Fasting glucose Number of metabolic syndrome symptoms
Ciliska, 1998	78 obese females	Randomized Trial Psychoeducation versus Education alone versus Wait-list control group <ul style="list-style-type: none"> 12-week treatment, no follow-up 	Body weight Systolic BP Diastolic BP
Hawks, 2005	32 females aged 18-22	Cross-Sectional Study Dichotomized individuals high and low in intuitive eating <ul style="list-style-type: none"> Measured with IES [60] 	BMI Body fat % Total cholesterol HDL cholesterol LDL cholesterol Total/HDL ratio Triglycerides Fasting glucose Cardiovascular risk
Hawley, 2008	225 obese/overweight women aged 25-68	Randomized Trial 3 non-dieting interventions <ul style="list-style-type: none"> Group-based non-dieting program with relaxation response training, Group-based non-dieting program, mail-delivered non-dieting program 10-week treatment, 2-year follow-up 	Body weight Systolic BP Diastolic BP

Table A.3 continued

Mellin, 1997	29 adults (93% female)	Open Trial Developmental skills training program for adult weight management <ul style="list-style-type: none"> • Skills include: strong nurturing, effective limits, body pride, good health, balanced eating, and mastery living • 18-week treatment, 2-year follow-up 	BMI Systolic BP Diastolic BP
Mensinger, 2016	80 obese women aged 30-45	Randomized Controlled Trial Weight-neutral versus Weight-loss groups <ul style="list-style-type: none"> • 6-month treatment, 2-year follow-up 	Body weight BMI Waist circumference Hip circumference Waist to hip ratio Systolic BP Diastolic BP Total cholesterol HDL cholesterol LDL cholesterol Triglycerides Fasting glucose
Rapoport, 2000	63 overweight/obese women, 25% non-white	Randomized Controlled Trial Modified cognitive-behavioral treatment with non-diet components versus standard cognitive-behavioral treatment <ul style="list-style-type: none"> • 10-week treatment, 1-year follow-up 	Body weight BMI Waist circumference Hip circumference Waist to hip ratio Systolic BP Diastolic BP Total cholesterol HDL cholesterol LDL cholesterol Triglycerides Fasting glucose
Steinhardt, 1999	357 adults, 50% female	Non-Randomized Trial Traditional Weight Control versus Diet Free Forever groups versus Nonvolunteer comparison group versus Control <ul style="list-style-type: none"> • 10-week treatment, 1-year follow-up 	Body weight Systolic BP Diastolic BP Total cholesterol

Table A.4

Summary of Results of Studies Evaluating Intuitive Eating's Impact on Health Indicators

Health Indicator	Improved	Not Related	Worse
Total	4	2	0
Cholesterol	Bacon 2002, Bacon 2005 Mensinger 2016 Rapoport 2000	Hawks 2005 Steinhardt 1999	
HDL	2	2	2
	Carroll 2007 Hawks 2005	Mensinger 2016 Rapoport 2000	Bacon 2002 Bacon 2005
LDL	4	1	0
	Bacon 2002 Bacon 2005 Mensinger 2016 Rapoport 2000	Hawks 2005	
Triglycerides	2	3	0
	Bacon 2002 Hawks 2005	Carroll 2007 Mensinger 2016 Rapoport 2000	
Systolic BP	5	4	0
	Bacon 2002 Bacon 2005 Hawley 2008 Mellin 1997 Rapoport 2000	Carroll 2007 Ciliska 1998 Mensinger 2016 Steinhardt 1999	
Diastolic BP	4	5	0
	Carroll 2007 Hawley 2008 Mellin 1997 Rapoport 2000	Bacon 2002 Bacon 2005 Ciliska 1998 Mensinger 2016 Steinhardt 1999	
Fasting Glucose	0	4	0
		Carroll 2007 Hawks 2005 Mensinger 2016 Rapoport 2000	
Fasting Insulin	0	0	0

Note: HDL = high-density lipoprotein, LDL = low-density lipoprotein, BP = blood pressure. Columns represent studies that showed improvements, no relationship, or worsening of health indicators in response to intuitive eating, respectively. Cells display how many studies found each of the outcomes for each health indicator.

One explanation for the mixed results is differences in study follow-up periods. It seems that the benefits of IE emerge over time; therefore, improvements may not be observed in studies with shorter follow-up periods [34]. The benefits of IE in regards to weight (i.e., maintained weight versus a weight loss and rebound) are most clearly observed in studies with longer follow-up periods, as well. Taken together, these findings suggest that – contrary to traditional dieting approaches – IE and the new weight-neutral paradigm may lead to both psychological and physical benefits that are maintained in the long-term.

Unknowns about IE that Will Be Addressed in the Proposed Study

Despite the above evidence of potential benefits, several gaps exist in the literature and are the focus of this thesis. There are many weaknesses in the body of literature evaluating the effects of intuitive eating on physical indicators of health. These include non-representative samples, inconsistent study designs, and a lack of foundational research. Nearly all of the current literature includes samples of obese, middle aged, white women. It is crucial to consider potential differences in the IE-health relationship based on sample characteristics, such as age, gender, race-ethnicity, socioeconomic status, and obesity severity. Further, there is no consistent intervention methodology, which could contribute to the lack of consistency in observed results. Lastly, there is a paucity of observational studies establishing a basic association between intuitive eating and physical health indicators that exists independently of BMI. In order to continue moving forward, the field must first take a step back to establish this baseline association and use that knowledge to design a systematic, consistent program based on the intuitive eating model. The objective of the current study is to observe the unique association of intuitive eating on physical health indicators, independent of weight status.

Current Study

Given the gaps in current literature, the proposed project makes a contribution by investigating the existence of a unique IE-health relationship in a diverse population. Specifically, this project will be the first to directly evaluate the relationship between IE and weight status, and determine whether IE is associated with health (i.e., cardiovascular factors, metabolic factors) independent of an individual's current BMI. These findings are important because IE could be a potential strategy to decrease disease risk for those with treatment-resistant obesity, and/or a potential adjunct for behavioral obesity treatments. The primary aims of this thesis are to a) determine whether IE is associated with greater overall health in a diverse population and b) evaluate whether this relationship remains after adjusting for BMI. The exploratory aim of this thesis is to determine whether there are differences in the relationships between IE, BMI, and health based on age, gender, race, and/or obesity status.

Hypotheses

Based on the current status of the literature (i.e., highly mixed) not many *a priori* hypotheses can be made. A critical evaluation of published studies reveals that well-designed studies most consistently report relationships of IE with total cholesterol and LDL cholesterol [32, 47, 48]. Therefore, it is hypothesized that IE will be negatively related to total cholesterol and LDL cholesterol. It is more difficult to make specific predictions surrounding the remaining dependent variables. For HDL cholesterol, systolic blood pressure, and diastolic blood pressure findings are split relatively equally, with some well-designed studies finding that blood pressure improves throughout a non-diet intervention and some finding no relationship between the variables [32, 42, 46-49, 52-54]. Alternatively, the current literature would suggest that IE is not related to triglycerides and fasting glucose [42, 46-48]. However, all of the studies evaluating the

impact of IE on HDL cholesterol, systolic and diastolic blood pressure, triglycerides, and fasting glucose were limited by small and/or uniform study samples. The present study will improve on these designs by including a larger sample of individuals from a broad range of ages, races, and weight categories and attempt to bring clarity to the literature. Lastly, the relationship between intuitive and fasting insulin has not yet been investigated. It is relevant to include fasting insulin in the present study because it is equally relevant to disease risks associated with obesity as the other dependent variables. Based on these limitations, no *a priori* hypotheses of IE's relation to HDL cholesterol, systolic blood pressure, diastolic blood pressure, fasting glucose, or fasting insulin are made.

In the current literature IE is consistently related to a decreased body mass index [51]. Therefore, we predict that BMI will be associated with each of the dependent variables. However, this will be the first study to directly evaluate the impact of IE on health indicators after adjusting for the effects of BMI. Consequently, we make no *a priori* hypothesis regarding the extent to which IE is uniquely associated with the dependent variables, above and beyond body mass index.

APPENDIX B

Intuitive Eating Scale-2

	Strongly Disagree	Disagree	Neutral	Agree	Strongly Agree
I try to avoid certain foods high in fat, carbohydrates, or calories.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I find myself eating when I'm feeling emotional (e.g., anxious, depressed, sad), even when I'm not physically hungry.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
If I am craving a certain food, I allow myself to have it.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I get mad at myself for eating something unhealthy.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I find myself eating when I am lonely, even when I'm not physically hungry.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I trust my body to tell me when to eat.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I trust my body to tell me what to eat.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I trust my body to tell me how much to eat.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I have forbidden foods that I don't allow myself to eat.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I use food to help me soothe my negative emotions.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I find myself eating when I am stressed out, even when I'm not physically hungry.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I am able to cope with my negative emotions (e.g., anxiety, sadness) without turning to food for comfort.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
When I am bored, I do NOT eat just for something to do.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
When I am lonely, I do NOT turn to food for comfort.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I find other ways to cope with stress and anxiety than by eating.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I allow myself to eat what food I desire at the moment.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I do NOT follow eating rules or dieting plans that dictate what, when, and/or how much to eat.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
Most of the time, I desire to eat nutritious foods.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I mostly eat foods that make my body perform efficiently (well).	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I mostly eat foods that give my body energy and stamina.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I rely on my hunger signals to tell me when to eat.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I rely on my fullness (satiety) signals to tell my when to stop eating.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
I trust my body to tell my when to stop eating.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

For each item, please check the answer that best characterizes your attitudes or behaviors.

VITA

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